CAPE OF GOOD HOPE.

COLONIAL SECRETARY'S MINISTERIAL DIVISION.

REPORT

OF THE

DIRECTOR

OF THE

COLONIAL BACTERIOLOGICAL INSTITUTE.

For the Year 1897.

Presented to both Houses of Parliament by command of His Excellency the Governor.

1898.

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CONTENTS.

PAGE
Annual Report 3
APPENDICES :—
1. Rinderpest 16
2. Morbid Anatomy of Rinderpest 29
3. Experimental Investigation of Rinderpest .. 37
4. The Question of the Relationship of Rinderpest to Variola 81
Historical Retrospect of Rinderpest 83
5. Horse Sickness—Investigation and Production of Curative Serum 105
6. Heart Water Investigation 116
7. Red Water or Texas Fever 120
8. Locust Fungus 123
9. Tables of Temperatures 127
10. Statement of Vaccines, &c., issued 135

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1897.

REPORT OF THE DIRECTOR OF THE COLONIAL BACTERIOLOGICAL INSTITUTE FOR THE YEAR 1897.

Bacteriological Institute,
Graham's Town,
10th February, 1898.

SIR,—I have the honour to submit to you my report of the work of this Institute for the year 1897.

RINDERPEST.

In the beginning of the year I was still stationed at Taungs, where my experiments were being conducted under very great disadvantage.

Early in May, Rinderpest having invaded the Colony, it was deemed advisable to proceed south with the object of finding some new situation where under better conditions further experimentation might be carried out. About the end of May I proceeded to Kimberley, at which place I was joined by my assistant, Mr. Robertson, who had been in the beginning of the year detached from my staff for the purpose of carrying out bile inoculation in Basutoland in company with Mr. Armstrong, one of the assistants of the Colonial Veterinary Surgeon.

After a short stay of several weeks at Kimberley I proceeded to Steynsburg for the purpose of forming a camp, where in addition to further experiment, I might be able to supply virulent blood for the purpose of starting various inoculating stations in the Midland and Eastern Provinces. After a few weeks, inoculation being already begun in the district of Alice, which had become previously infected, I proceeded to break up the camp at Steynsburg and to carry out further work at my headquarters in Graham's Town.

The line of work which I had for the most part followed out at Taungs was concerned with the blood of Rinderpest animals, with regard to which certain peculiar facts were noted.

(1.) It was found that such blood lost all its virulence after being kept for nine days (the mean daily temperature having been about 100 degrees Fahrenheit in the shade).

(2.) Where the blood after keeping for six days was used to inoculate a healthy animal, the malady produced was modified in character and the animal recovered.

(3.) Where the blood was used within five days of its withdrawal from the body, virulent Rinderpest was produced.

Thus from a consideration of Clause (2) it will be seen that theoretically a method has been devised for the induction of an attenuated form of Rinderpest; but as it was obviously impossible to hit off the exact period during which such a condition would be produced, the result in the field would be either to fail in setting up the disease at all or to set up virulent Rinderpest unless, by hitting off the exact period, the attenuated form had been produced.

These observations have been amply corroborated in the field, for as I have been in the habit of sending virulent blood to the various stations, it has happened, where the blood has been overcarried on the railway or by other means so delayed, that it was not used till after the sixth day, no Rinderpest was produced or only in a modified form, from which the animals recovered.

This has occurred in the case of blood both defibrinated and kept in citrate solution.

In the case of blood which had been kept for such a time as admitted of the virulence being lost, I discovered that the inoculation of 30 c.c. of such blood, to which glycerine had been added, into a healthy animal, produced a measure of immunity which was sufficient usually to withstand the injection into the same animal of 0·2 c.c. of virulent blood at a later date. The difficulty, however, of procuring blood in the field was a barrier to the ready application of this method, and as Dr. Koch had already discovered the immunizing property of fresh Rinderpest bile, and when reports began to come in relative to the occurrence of Rinderpest in herds soon after such inoculation, I thought it advisable to determine whether bile treated by glycerine could not serve to induce passive immunity. My experiments in this direction shewed that if bile is taken from the gall bladder of an animal which has died from the disease, provided that it is not highly putrid, it can be glycerinated and used, without risk, to produce a weak immunity.

The best bile for this purpose is that which is taken from a dead animal which has been infected spontaneously or by smearing the nose and mouth with virus, or by means of a subcutaneous injection of a very small dose of blood.

Bile from naturally or spontaneously infected animals is best of all, next to which comes that which is produced by nasal infection.

In this method nearly all biles serve for glycerination, provided they are not thin and pale in colour nor septic.

In practice two parts of bile are mixed with one part of glycerine, and the mixture is kept for eight days previous to use. The dose adopted is 15 c.c. for calves, 20 c.c. for medium-sized cattle, and 25 c.c. for older and heavy animals.

Ten days after inoculation with such bile, 0·2 c.c. of virulent blood is to be injected subcutaneously.

Where the bile has been of the proper character and the blood taken from an animal at the height of the disease and suffering from *no other complication*, the result has been that a very small percentage at most in the herds have died, while in very numerous cases no deaths whatever have followed.

The immunity so produced is, however, of an uncertain duration, because we have recognized that the permanence of the immunity, as I have stated in my first report on this disease, is in proportion to the amount of illness or reaction produced. I therefore advised that the blood inoculation should be followed ten days later by a second and larger injection of virulent blood. The result of this has been that in herds where no visible reaction followed the first inoculation, a measure of sickness and mortality succeeded on the second, but in general the results have been extremely good.

In herds which have been already infected before inoculation, the use of good glycerinated bile has in many instances arrested the spread, and the herds have been immunized with but a small percentage of loss.

Glycerinated bile of itself, if repeated at intervals, gives a fair amount of protection against spontaneous infection, and some transport riders are using this with satisfactory results.

No method, however, gives any degree of permanent immunity unless a febrile reaction has been sustained by the animals inoculated.

Dr Koch's Rinderpest bile method was certainly the first real advance towards immunizing cattle, and, despite the fact that Rinderpest has frequently followed in its train, I am emphatically of opinion that it has been the means of protecting, at all events for a time, hundreds of thousands of cattle, and has further been of service in producing large numbers of "salted" cattle, which have furnished the necessary means to the determination of the serum method. I am convinced, where the bile is taken from animals dying of spontaneous infection or infection produced by smearing the nose and mouth, and where such bile is selected in accordance with the directions, that a very high degree of immunity can be conferred with but a slight percentage of mortality.

For my own part I prefer to see a slight mortality follow, because experience has shewn me that in almost every herd there are a few animals that are more highly susceptible to Rinderpest than others, and the mortality induced serves to give an index as to the febrile reaction that has occurred in the mass of the herd inoculated.

Further, the use of bile has this advantage, that where immunity breaks at a later date it does so irregularly, only one or two at a time becoming infected, and as these are more amenable to serum than animals not formerly inoculated, they are easily dealt with.

SERUM.

Dr. Koch shewed that the serum from a "salted" animal had an immunizing property, but the immunity produced was not of long duration. Drs. Danyz and Bordet shewed that the immunizing strength of the serum was most variable in different animals, and that it could be raised by subsequent injections of virulent blood. They also devised the ingenious method of infecting the animals in a manner comparable to spontaneous infection and inoculating them with serum. By this means they succeeded in inducing a modified form of the disease with but a small percentage of deaths.

Drs. Turner and Kolle have sought to reduce the dose of serum by repeated injections of increasing doses of Rinderpest blood, and by using a serum standardized to the antagonistic action of Rinderpest blood, they have succeeded in arriving at the induction of a modified form in using one cubic centimetre of virulent blood on one side and 20 cubic centimetres of serum on the other. The objection, however, to blood infection is, firstly, that should spontaneous infection occur before or during the period of the modified reaction, then Rinderpest appears in great virulence. Where the animals are under close observation this does not so much matter if plenty of serum is procurable, but it certainly is of a great disadvantage in herds in the open, and especially on large farms where it is a serious difficulty to bring the cattle together. Secondly, subcutaneous blood inoculation is distinctly dangerous in this country, because the blood of animals from red-water areas can and does produce red-water in susceptible cattle, even although the animals from which the blood is taken do not shew any indication of illness. My experiments (see section on Texas Fever) shew that animals partially immune to red-water can be infected with it without giving rise to sickness, and the blood of these animals three weeks later if inoculated into clean susceptible animals will set up Texas Fever. Moreover, the occurrence of red urine as a symptom does not always follow, and, where the immunity is partial, never does follow.

In such cases the parasite in the blood is greatly modified in form, so that instead of the double pear-shaped microbe there appear round globular elements which strikingly conform to the parasites described by Drs. Turner and Kolle as having occurred among cattle at the Kimberley compound and at Robben Island. In such cases an enormous amount of blood destruction occurs, and the blood of the animals is watery and translucent, and either does not coagulate, or at best in a very imperfect manner.

To obviate this danger I have had recourse to a method which is after the manner of Drs. Danyz and Bordet. I mix virulent blood with water in the proportion of one of the former to thirty of the latter. The animals are drenched with one third of a quart bottle full, and after three days receive 50 to 100 c.c. (according to its strength) of fortified serum. One then waits till the temperature begins to rise, and a further dose of serum is then given. This method has worked very well in my hands, and with a little expenditure of serum is comparatively safe. The advantage is that red-water is not so induced, and since the nose and mouth are infected by this means, one has no fear of spontaneous infection.

In using serum it has to be borne in mind that the strength is variable unless it has been taken from animals fully fortified. The serum of the blood of an animal which has recovered from a modified or mild attack is not usually very strong, but where an animal has recovered from a mild attack induced by blood inoculation, and is then infected by the nose and mouth a second, severe, but not usually fatal attack follows, and when recovery has succeeded the serum always possesses a high degree of strength. Thirty c.c. of such serum given to an animal 24 to 36 hours after its nose and mouth has been abundantly smeared with virulent nasal mucus usually entirely arrests the progress of the disease, and no temperature reaction occurs.

It is evident from this that to give serum at the same time as the nose and mouth is infected is useless unless the serum is of a very weak character, otherwise the infection is nullified, and the animal eight or ten days later is accessible to infection.

I have also found that the serum from the blood of an animal suffering from, and in the later stages of Rinderpest, has immunizing properties on which, probably, depend the results I have formerly obtained with glycerinated blood. This power, however, is weak, and is associated with very little, if any, curative power.

Curative power is seen at its best in an animal which has been salted from a second attack of Rinderpest almost immediately following upon a mild one,

In the case of fortified serum I prefer to fortify by using doses of virulent blood up to 1,000 c.c., which should be given on two or more occasions, then after a week's rest 100 c.c. of virulent blood is injected into the jugular vein, and the animal left for at least ten days previous to bleeding it. By this means a powerful immunizing and curative serum is obtained. The blood used for intravenous injection should be at its maximum of virulence, and the virulence may, if necessary, be exalted by passing through Cape sheep as Dr. Koch shewed.

In the case of spontaneously infected cattle, these do not respond to the action of serum so readily as those which have formerly been bile inoculated, but I have found in the case of these that if the animal is bled freely (about six quarts) and then inoculated with 100 c.c. of fortified serum that it usually recovers.

In using serum to the best advantage the disease should be produced in the presence of serum, and then cured by it, and where the serum in the first instance arrests the production of fever, a second inoculation of virus should be made not later than ten days after the first, and the process repeated.

In the appendix I have dealt at considerable length with the experimental work which I have carried out in regard to Rinderpest and its morbid anatomy.

This disease has been for the greater part of the civilized world a menace, and for many countries a bitter experience I have, therefore, also included a short historical retrospect of the subject, bringing it as far as possible up to the date of the invasion of this country.

From time to time it has been re-affirmed that Rinderpest was related to Variola (Small-pox).

In the appendix will be found a section dealing with this point, and I believe the experiments therein alluded to will be accepted as shewing that in no way further than a slight similarity in certain pathological lesions are the two conditions related.

RED-WATER, OR TEXAS FEVER.

In the appendix will be found a section dealing with this disease, but only as it has come into relation with Rinderpest inoculation.

The experiments show the danger of using the blood of animals that have been living on so-called red-water areas, and that are believed to be immune to that disease.

Moreover, it would seem that by the inoculation of such cattle with virulent red-water blood, a condition is produced in which the blood becomes thin and watery, and the specific form of the parasite is changed to that of spherical bodies which are mostly included within the red blood corpuscles. Where such a condition is produced in an animal from a red-water area only a slight irregularity of temperature is produced (the animal being already partially immune) but occurring in such an animal infected with Rinderpest the result is usually attended with death. In such cases the peculiar thin translucent blood is sufficient to arouse suspicion.

HORSE-SICKNESS.

Two salted horses of the number which were sent from the Institute to Bechuanaland about two years ago, and since exposed day and night during a very severe season of horse-sickness, were returned to me at my request These animals have been re-inoculated with excessively large doses of the most virulent blood of animals dying of horse-sickness, but have shown themselves to be absolutely immune.

After a period of treatment by the injection of large doses of virulent blood, serum has been obtained which has a decided curative action upon animals in which the disease has been produced by inoculation.

In the appendix will be found a comparative table of temperatures shewing the progress of the disease till death in an animal infected and not treated with serum, while there will also be seen the effect, ending in recovery, in an animal infected and treated with serum. Unfortunately the amount of serum required is rather considerable, and in order to provide sufficient for inoculation on an extended scale a large number of horses would be required. To this end I am now "salting" horses and am endeavouring at the same time to determine if it is possible to obtain equally successful results with a less amount of serum. The serum of "salted" horses has of itself almost no value, while that of animals to which large doses of virus have been given has to be used with caution.

In the absence of any simpler means I believe that the use of this method might come into use as a means of "salting" horses against this terribly fatal malady.

HEART-WATER.

This disease, which may be looked upon as having arrested the wool production in many parts of the Eastern Province, has been believed to be due to a depreciation in the pasture lands.

From time to time the attention of the Veterinary Staff has been directed to this malady, but so far with little avail. Experiments which had been made for the purpose of transmitting infection from sick to sound animals were attended with negative results.

I have on various occasions, as circumstances permitted me, attempted to determine whether or not it was of the nature of an infective disease, but the advent of Rinderpest, which called for the use of all available staff, precluded my carrying out further research at that time. I had, however, satisfied myself that it was communicable from sheep to sheep and from goat to goat.

This year I was enabled to procure a typical case in a goat, and from the blood of this animal in the Institute I infected goats and sheep which died of typical heart-water. I also used the bile of the animal dying to inoculate others—a goat and a sheep—of which both, after a period of incubation which lasted eight days and corresponded with the blood inoculation, had an onset of high fever which ended in recovery.

The onset of fever in the goats is more marked than in sheep. In the former there is a very sharp rise with a high ascent, followed by a descent almost to normal from whence a gradually rising temperature is seen. In the sheep the chart is very irregular.

So far as my experiments go they admit of the hope that a modified form of the disease can be given by inoculation with bile, and I am now determining the smallest dose of bile which will serve, as, there being only about 50 c.c. in the gall bladder, it becomes a matter of great importance to economize.

Should this method succeed it would be necessary to inoculate during the season when this disease is not present in the veld, and it would be necessary to prepare virus for inoculation during the winter or early spring.

LOCUST FUNGUS.

During the year extremely good results have been reported to me as accruing from the use of this fungus, which has now been supplied to Zululand, Natal, Free State, Transvaal, Basutoland, East Africa, Rhodesia, Northern Africa, Buenos Ayres, Australia, United States of America and India.

An account of the first discovery of the fungus will be found in the appendix.

Recent information shews that another fungus has been seen at Buenos Ayres which is stated to act well in dry and hot weather. I am endeavouring to obtain some of this fungus or infected locusts, and should it be proved serviceable I shall endeavour to propagate it here.

VACCINE LYMPH.

The preparation of vaccine lymph has been carried on during the past year under great difficulty. During the first half of the year I was absent from head-quarters and engaged on Rinderpest work. During the latter half of the year great difficulty has been and is still being experienced in procuring suitable calves for vaccination, as owing to the advent of Rinderpest the farmers were averse to permitting us to obtain their calves. Now the difficulty is caused by the fact that since many cows under inoculation have cast their calves, there is a great scarcity, while of those born many are poor, stunted and weakly. This state of affairs, occurring, more especially during the height of the warm season, when under the best conditions the lymph from calves is less efficacious than during the colder weather, has made the matter of maintaining the supply difficult in the extreme.

STATISTICAL.

During the year 43,297 tubes of vaccine lymph have been issued, representing a value of £3,248, 70 tubes of lung sick virus and eight needles to the value of £6 3s. 6d., 21 bottles of diphtheria antitoxin and 31 syringes to the value of £20 11s. 6d., 2,559 tubes of locust fungus to the value of £40 12s. 4d., 178 tubes of sponziekte vaccine and five syringes to the value of £12 8s., six bottles of mallein to the value of 15s., 1,444 tubes of Miltziekte vaccine and 56 syringes to the value of £203 11s. 6d. During the last four months of the year 555,300 cubic centimetres of Rinderpest serum and syringes were sent out to the value of £1,991 4s. 8d.

The number of analyses and examinations made were as follows :—

Bacteriological—	
Diphtheria	5
Phthisis	28
Urinary	14
Pathological —	
Tumours, &c.	16
Chemical—	
Water	6
Viscera	1

CONCLUSION.

In conclusion I have to express my grateful acknowledgment of the assistance which has been rendered me by my assistants Dr. Purvis, Mr. Robertson, M.R. C.V.S., and my clerical assistant Mr. Hedley, B.A., also the service which has been temporarily given me on Rinderpest duty by Dr. George Robertson.

To my colleague, Mr. Duncan Hutchem, C.V.S., I desire to tender my cordial thanks for his kind co-operation wherever such has been required.

The work of the Institute having been considerably augmented I have to express a hope that the alterations now so necessary and already under consideration may meet with approval.

I have the honour to be, Sir,

Your obedient servant,

ALEXANDER EDINGTON, M.B.,

Director.

APPENDIX

TO

ANNUAL REPORT FOR 1897.

Appendix I.—Rinderpest.

Definition.—According to Gamgee, the plague among horned stock which constantly pervades Russia, and especially steppe lands, is a specific, malignant, and highly contagious fever, known to us only as the result of direct or indirect, communication from sick to healthy animals. It is entirely *sui generis*, and never originates spontaneously beyond the Russian frontiers; within which it is probably not generated at any time *de novo*, but is kept up by constant reproduction, after the manner of other contagious maladies. It is essentially a bovine disease, though it may be communicated to goats, sheep, deer, antelopes, gazelles, aurocks. zebras, and even the peccari, never attacking any animal more than once. It is characterised by a period of incubation which does not exceed ten days; by fever heat which precedes all other symptoms; redness of all visible mucus membranes, seen early and in a marked manner in the vagina of cows; sometimes delirium, muscular twitchings and fever shivers; discharges from eyes and nose; normal secretions checked or suppressed; abdominal pain with constipation or diarrhœa; scaly eruption on back and loins, and in some epidemics a characteristic eruption on the inside of the thighs and on the mammæ; there is tendency to early putrefaction manifested by the foetor of breath and discharges and emphysematous swellings in various parts of the body before death; after death rapid decomposition sets in. The majority of animals seized with the cattle plague, as a rule, die wherever the disease appears, except among Russian herds. No specific antidote has been discovered for the poison.

From the third report of the "Commissioners appointed to enquire into the origin and nature of the Cattle Plague (London, 1866)" we learn "that enquiries set on foot, first by Professor Gamgee and then by Dr. Sanderson" established the important fact that a rise of temperature precedes any other symptom. They found that within a period ranging from 36 to 48 hours after an animal had taken ill with the Cattle Plague by inoculation that the normal temperature rose to 104° Fahr. They found that this took place at a time when the animal appeared in no wise ill, and they concluded therefore that the incubation period was considerably shorter than had been supposed. This discovery they deemed of great practical importance, as it might and ought to lead to an earlier separation of the sick from sound animals, and might admit also of a shortening of the period of quarantine. Two days after the rise of temperature was observed they were able to notice the onset of another symptom, namely a peculiar condition of, or an eruption on, the lining membrane of the mouth. This eruption at first sight shewed a close resemblance to that found in foot and mouth disease, but was readily distinguishable by a practised eye. Dr. Sanderson found it in every case seen by him, and he was able to identify the disease from this sign alone. It was, however, in some rare cases, absent. Almost coincidentally with the advent of this symptom there appeared a peculiar and distinctive condition of the mucous membrane of the vagina.

About a day after the appearance of the eruption, or about 72 hours after the rise of temperature, the animal was observed to have less appetite than usual and to ruminate irregularly. About a day later, or 110 hours after the initial rise of temperature, the animal was clearly ill; then ensued the urgent symptoms, the drooping head, the hanging ears, the distressed look, the failing pulse, the oppressed breathing, the discharges from the eyes, nose and mouth, the eruption of the skin, the foetid breath, and other well-known signs of the disease. During the sixth day the contractile force of the heart diminished, the pulse became small and thready, the respiratory movements became modified, and the animal sometimes shewed such weakness in the limbs as to raise the suspicion that some special paralytic affection of the spinal nerves had existed. The temperature now began to fall with coincident signs of a great diminution of the normal chemical changes of the body. Death usually occurred on the following or seventh day from the date of the first or initial rise of temperature.

From this as a typical course of the malady, there occurred considerable deviation both in point of time with regard to death and in regard to severity and sequence of the individual symptoms.

SYMPTOMS OF RINDERPEST OR CATTLE PLAGUE AS SEEN IN SOUTH AFRICA.

My observations of Rinderpest, as it occurs spontaneously, *e.g.*, natural infection, have been made among the numerous herds of cattle belonging to the natives of the Reserve in Bechuanaland and the native Reserves in the Western portion of the South African Republic. The stations also at which my assistant and myself have conducted experiments in various parts of South Africa, have admitted of valuable observation; but of all of these, the thousands of cattle in the native Reserve at Taungs and Phokwani, which died of Rinderpest, unmodified by treatment or inoculation, best admitted of careful and prolonged observation being made. From experimental inoculation made by placing small quantities of the nasal mucous from affected on the nasal membrane of healthy cattle, I was able to determine that the first rise of temperature occurred at a period which varied between 51 and 72 hours after inoculation. The rapidity of the onset, as also the severity of the subsequent symptomatic phenomena, were dependent upon—firstly, the virulence of the case from which the virus was obtained; secondly, upon the freshness of the virus (where the virus was kept exposed for 24 hours to air the resulting disease was greatly modified); thirdly, upon the amount of material used for inoculation (where a small amount was implanted on the nasal membrane with the finger, the incubation period was longer; where freely rubbed over the nose with a well-soaked sponge, the period was as short as in any case of inoculation hypodermically with virulent blood).

About a day after the rise of temperature which followed inoculation had occurred, it was seen that the muzzle was somewhat dry, and the animal less inclined for food than usual (animals confined and fed on dry forage). A little later the animal refused or only nibbled his food in the early morning, but sometimes fed later on in the day. About 36 to 48 hours after the rise of temperature, the mucus membrane of the inner portion of the eye was injected and soon presented a vivid pink colour. In very many instances this was one of the earliest visible signs of the disease. Just about this period a slight watery discharge from the eyes may be noticed. In the high veld of Bechuanaland where dust storms are common such a discharge is frequently to be observed in healthy cattle, but observations extending over thousands of cattle admit of any fallacy in this observation being eliminated. Very soon after the onset of this secretion had been noticed, it was seen that the normal nasal discharge was much augmented in volume and changed in character. The healthy discharge is somewhat viscous from the presence of mucus, so that it tends to drip in fine threads; but when the plague secretion has begun to make its presence manifest the secretion becomes much more watery in character and drops on the ground. Very soon the skin of the upper lip and muzzle, where uncovered by secretion, became unnaturally dry, thickened and cracked, due to the action of the abnormal secretion and saliva exuded from the mouth. At this period the appearance and attitude is indicative of sickness; the animal if in the open leaves the herd and if water is near will stay by it; the ears hang forward, the head droops, and flies settle around the head without much effort being made by the animal to dislodge them. Still later diarrhœa sets in, and very soon after it has started assumes a violent form with the emission of a characteristic stench. By this time the attitude of the animal will have become most characteristic; it stands with its four feet stretched slightly forward, as if to take every advantage of mechanical support and relieve muscular effort. The head droops, eyes, nose, and mouth discharging; the ears droop heavily, the back is arched, the skin is hide bound, while the constant purging often freely mixed with blood, the short painful groan or grunt, and the general helpless look make up a picture of utter hopelessness. Where the animal lies down it is common to find the head turned towards and the nose tightly pressed against the flank, an appearance indicative of intestinal irritation. The avidity for water, partly perhaps due to the heat of the climate, largely I consider due to the fever and characteristic of the plague, is a marked feature.

At this time the river which passes through the reserve was nearly dried up, and in the deep pools which were left it was common to find numbers of dead animals.

Among animals which have been inoculated subcutaneously with virulent blood, the symptoms are considerably modified. The secretions from the eyes, nose, and mouth are much less marked, and in some cases almost absent, while conversely the general implication of the intestinal tract is more evident.

Death occurs from the tenth to the fourteenth day following upon inoculation with nasal mucus.

The duration of the period of incubation following upon inoculation with virulent blood depends upon, first, the virulence of the blood used, and secondly upon the amount. In my experience the first onset was most usually determined in the evening, in cases where the temperatures were observed thrice daily, and the average period of onset would be about 60 to 80 hours subsequent to inoculation, or considering the day of inoculation as the first day, the first rise of temperature would occur on the evening of the third day as the earliest period, and the evening of the sixth as the latest.

This must be understood as applying to inoculation with 2 to 3 cubic centimetres of virulent blood obtained from animals suffering from the disease acquired spontaneously. If one maintains a strain of virulent blood by transfer from one animal to another, and the blood be constantly selected at an early stage of the sickness, then a slight shortening of the period of incubation is discernable, so that the rise of temperature may occur within 60 hours of the moment of inoculation. In the report of the English Commissioners they speak of the temperature rising within 36 to 48 hours after inoculation. The earliest rises I have encountered in my experiences was 51 hours after inoculation with nasal mucus, and 54 hours after inoculation with virulent blood injected subcutaneously.

SUMMARY OF THE SYMPTOMS OF RINDERPEST.

The earliest symptom, namely a rise of the temperature, can only be determined by the thermometer, and it is necessary in making use of this aid to take the hour of the day and the atmospheric conditions into consideration. The morning temperature in health is lower than the mid-day, and the evening is usually the highest.

The normal temperature may be 99° Fahr. to 100° Fahr. about 7 a.m., 100° to 101° at mid-day, and 101° to 102° in the evening about 6 o'clock. Slight variations may occur, and about an hour after drinking water it is usual to see a fall of half or one degree.

A cold day may cause a slight oscillation of the temperature of a healthy animal but during disease such oscillations are much more accentuated.

It is convenient to divide the phenomena occurring after the temperature has risen, into three stages.

First Stage.—The animal is dull and may have a shivering fit, the coat is staring, the lining of the mouth is redder than normal, the ears droop and there may be slight cough, the bowels are somewhat constipated, and the foeces will be seen to be coated over with slimy mucus, the urine is high coloured. The appetite is now capricious or lost, great thirst is evidenced, twitching is seen of the superficial muscles of the back, shoulders, or hind quarters; rumination is irregular and slow, grinding of the teeth becomes almost constant, the animal yawns, tenderness is present over the spine, the temperature is high and the pulse increased.

Second Stage.—The horns, ears, head, and other parts of the body are variable in temperature, now hot now cold; appetite is lost, rumination ceases, respiration is quickened, a slight watery discharge is seen from the eyes, the mucous membrane at inner corners is injected and of a pink colour, tenderness of the spine is most marked, when lying down the head is commonly turned in towards the flank, temperature is very high (105°-108°) thirst great, twitching of superficial muscles more marked, pulse is quick and also frequently irregular, the mucous membrane of gums and sides of mouth very red, tongue is furred and papillæ very red, bowels still constipated, dung coated with mucous and streaks of blood. Mucous membrane of rectum and vagina dry and of a dusky red colour.

Third Stage.—Eyes, nostrils and mouth discharging profusely, breath has a very offensive odour; there may be excoriations of gums, corners of mouth, buccal mucous membrane, roof and floor of mouth, and of tongue. Diarrhœa now begins, and the hard foeces, mucus coated and blood streaked, give place to dirty watery foeces which later consist of mucus flakes and altered fibrin with blood. The odour is offensive and characteristic.

Protraction is very great, thirst continues, with great difficulty in swallowing; coughing. Animal lies down, moans or grunts, liquid foeces are passed involuntarily, pulse is very weak and thready. If the temperature suddenly falls below normal it is an almost invariable sign of the near approach of death. In other parts of the world a cutaneous eruption frequently appears, and when so it is said that dysenteric symptoms do not usually prevail, that the disease is mild in type, and that such phenomena are associated with the hot season of the year.

The symptoms described are not always constant, either in sequence, character, or severity.

The duration of the disease, from the moment of the rise of temperature till death is usually five to twelve days, the average being about eight. One of the earliest signs of recovery is seen when the dirty liquid foeces become tinged with bile.

In some cases the disease is mild in type, and in such cases it is common to notice early in the progress of the malady a collection of pus or muco pus at the inner corners of the eyes. This symptom has been so commonly found in cases which went on to subsequent recovery that we have become accustomed to regard its advent as indicative of benignity.

As to the causes which lead to the production of the train of symptoms which have been described, all are agreed that certain morbific particles first of all gain access to the healthy body, and to the subsequent operation of these the various alterations from health must be ascribed. The specific nature of these particles cannot yet be held as ascertained. In the blood, in a condition of health, are to be found bodies which are variously named as "blood plates," *(blût plattchen* " and "*hæmatoblasts).* In disease, especially of an infective character, these are much more numerous, and in Rinderpest it is common to find them in enormous numbers in the early and later stages of the disease. It is possible that the specific cause of Rinderpest may have some resemblance to these bodies, and, if so, the presence of the latter would tend to make observations more difficult. The closest observation with the highest powers of the microscope has so far failed in identifying any particular microbe as being the cause of the disease, although it has been found that among cattle in one area similar organisms, of the nature of secondary infections, have existed in the blood of numbers of the animals.

Among the earlier cases investigated at Taungs there occurred a peculiar organism of a distinctly polymorphic type of a character which does not admit of its exact place among bacteria being recognised. In another series, which began with a spontaneous case and transferred itself through the blood of several animals subsequently inoculated, there occurred a streptococcus which has been isolated as a pure culture. These are all undoubtedly of the nature of secondary infections, which in the first instance had gained access to the blood either from breaches in the continuity of the mucous surfaces of the bowel or throat, or from invasion through the tonsils. In the case of the streptococcus referred to, the route by which entrance had been effected was undoubtedly by the latter channel, as examination of the glands named shewed the presence of the microbe deep in the substance of the gland tissue, and particularly in relation to pin point abcesses which no doubt owed their origin to the specific action of the microbe in question. In the blood of some other cases in the Taungs Reserve we found a peculiar body present in many of the red blood corpuscles in the form of a frequently paired microbe, which has been identified in America as the cause of Texas Fever.

It is noteworthy, however, that some of the cases in which this microbe occurred gave no indication before death of the presence of this disease. After death, the only condition found as suggestive of the malady was the great engorgement of the bilary canaliculi with bile and the mahogany-like colour of the liver and its fluorescent appearance when viewed by reflected light.

The urine contained in the bladder was scarcely ever bloody, but in a few cases minute brilliant red points were found on the mucus membrane which are commonly found in this disease. I also observed that if clean cattle sent from the Kimberley district to the station were kept there for about a month that the microbe in question could be found in the blood It never occurred in the blood of any experimental animals which died there if kept within that period. The characteristic symptoms of Red-water, notably the staggering gait, red urine, and watery condition of the blood was not observed in any of our cattle, nor, indeed, was it seen in any of the spontaneous cases of Rinderpest among the native cattle in which the microbe was afterwards identified.

It is well known that the cattle in districts which have been long infected with Red-water become "salted" to the disease, and their progeny born in the area are more or less immune, so that I infer either that the native cattle in which I identified the microbe were salted cattle whose immunity had been upset by the Rinderpest, or that the microbe may gain access to the blood of immune cattle without setting up the train of symptoms commonly ending in death, which are collectively known to us as indicative of Red-water.

The specific microbe which gives rise to Rinderpest multiplies within the body in an incredibly short space of time, but my observations lead me to believe that it is difficult to say at what time an infected animal can infect others. In the case of spontaneous infection spreading among a herd I am strongly of opinion that the disease spreads by the animals licking one another. As the smallest amount of fresh nasal mucus from an infected animal when placed high up in the nose of a healthy one seems in the most certain manner to infect the animal, it would seem as if this situation was one of natural selection, and it is very possible, therefore, that animals infected by this route may transmit the disease to others previous to the time when the fever shows itself in them.

In the epidemic which occurred in England in 1865 there were constantly observed almost everywhere in the body, but especially in relation to the mucous surfaces and skin, about the third or fourth day, local congestions which varied in size and intensity.

In South Africa these phenomena have not been conspicuous, and indeed although I have made a careful search I have failed to find any eruptive conditions of the skin. In the mouth occasionally one finds the following:—(1) Slightly raised vesicular points which are probably related to (2) flattened irregular discoloured points which appear to originate in small submucous extravasations of blood and these in turn probably give origin to (3) shallow ulcers with irregular sharply cut margins, the latter being usually injected and contrasting with the greyish central area. In the later stages the floors of the ulcers are blood-stained. Such appearances are sometimes found on the buccal surface, on lower surface of the tongue and more commonly on the mucous membrane of the roof of the mouth.

One of the earliest changes is a congestion of the mucous membrane of the fourth stomach, and as this especially affects that part which is involved in the digestion of food it results in a failing or capricious appetite; rumination comes to a standstill and it thus happens that the stomach is filled with undigested food.

At the post-mortem examinations I have frequently found that the mucous membrane of portions of the intestine was in parts absolutely destroyed; the functions of absorption by the villi were in those parts rendered impossible, so that the food necessary to the animals existence must have been almost entirely wanting.

In some cases one has found that reparative effort was at work even while destructive processes were in their earlier stages at other parts but obviously reparative effort is limited to such portions as have not become entirely destroyed. In many cases the mucous membrane has been entirely removed and the intestine becoming transparent allows of its contents being seen through the walls.

Such contents frequently consist of casts or sloughs of the mucous membrane of bowel, mixed up with a badly smelling fibrinous material, irregularly mixed with the watery contents. In some cases a short cough or grunt is noticeable late on in the history of the disease, and after death in spontaneous cases it is common to find especially at the apices and free borders of the lungs considerable interlobular emphysema. Dr. Koch and myself tried to determine in the veld the nature of the gas contained in the spaces thus caused, but so far as we, under the circumstances, could judge, it consisted merely of air. Emphysema also is sometimes found in the cellular tissue of the back and posterior quarters.

One condition, however, which, from its intensity and constant appearance, would seem to be peculiar and to which I have formerly drawn attention is the marvellous enlargement of the lymphatic glands throughout the body, and especially of the intestine, particularly in the mesentry supporting the small intestine. The glands may be several inches in length presenting the appearance of sausage-like bodies. The colour is greyish, and on section the outer portion or cortex is whitish, but becomes dark clay-coloured in the medullary tissue. In some cases the smaller glands which are in the earlier stage of enlargement are soft, black and edematous, exuding considerable fluid when cut into, while the larger and older are usually somewhat firm in texture.

Animals which have recovered from the disease after a very severe attack very commonly present a peculiarity with reference to their blood, which coagulates very slowly and often imperfectly. I am accordingly impressed with the idea that this peculiarity has some relation to the enlargement of the lymphatic glands, seeing that this condition remains for some time after recovery.

From a general consideration of the *post-mortem* phenomena found in animals which have suffered from Rinderpest it is evident that there is more or less throughout the whole body a tendency to local congestions. With such a condition acting, it is not to be wondered at that such extravasations of blood are common, and since it is obvious, owing to the febrile state and the intense hyperplasia of the lymphatic glands, that the normal functions of absorption are probably interfered with, these extravasations must act destructively, firstly, in causing pressure among important glandular structures; and secondly, in shutting off superjacent portions of tissue from their proper nutrient supply. In the latter case it is more than possible that numerous areas of highly developed mucous membrane lying superjacent to extravasations will become the subjects of atrophic and necrotic processes, and the tendency to local congestions being more or less general, it is obvious that extravasations will occur more or less in obedience to physical laws.

Such a consideration of the phenomena acting in this disease serves largely to explain the conditions that are found on making *post-mortem* examinations of animals dying of Rinderpest.

The extravasations of blood so commonly and widely diffused throughout the body may be recognised as consisting of minute petechiæ or points, which in the larger discolourations are entirely or in process of becoming merged together.

These are found more or less throughout the mucous and some serous membranes. On the visceral layer of the pericardium such extravasations are common, and are usually situated near the apex of the heart in and on either side of the interventricular furrow, where the subserous tissue is largely areolar, and hence looser in texture. Within the heart also extravasations are common in the subserous tissue of the endocardium, and are commonly in close relation to the attachments of the papillary muscles. Throughout the whole intestine congestion is a marked feature. Extravasations of blood occurs irregularly; and ulceration, while being common in certain areas, is rare in others. Thus the œsophagus and rectum are less liable to extravasation and ulceration, probably because of the special constitution of these parts in which we find a superficial epithelial layer that is stronger than that of other parts of the gastro-intestinal tract, while the subjacent filamentous connective tissue forms a fairly complete framework that interlacing in all directions, lends strength to the tissue.

APPENDIX 2.—MORBID ANATOMY OF RINDERPEST.

The Skin.—In animals affected by the Rinderpest as seen in South Africa I have failed to determine any cutaneous lesions of a specific type.

In some cases the skin of the flanks shews, in relation to the hair follicles on the outer surface, a slight encrustation, and among this small reddish points may be seen. This condition, however, is not peculiar to Rinderpest and seems to be caused by the action of parasites. So far as my experience goes, specific lesions of the skin are very rare in this country. I would not absolutely deny their existence, because the condition of the skin in cattle is such in this country, owing to the action of ticks and other parasites, that it would be difficult to say in some cases whether areas of irritation were solely due to external parasitic life or were in part the expression of a specific constitutional disease. It is, however, to be also observed that a slight encrustation of the skin due to excess of sebacious matter is commonly found among healthy cattle. Careful examination shews that it is associated with an irritable condition of the sebacious glands and as a similar condition has been described as associated with Rinderpest in Europe, it is a very difficult thing to say whether Rinderpest has not a similar tendency among the Colonial cattle. I have not met with pappular or vesicular conditions on the skin of Rinderpest cattle.

The skin covering the upper lip is very commonly dry, hardened, and thickened, and in some cases the thickened epidermis is cracked, giving rise to a condition nearly allied to true eczema. This is, however, due to the discharge from the nose setting up irritation in a manner similar to that obtaining in a human being who is the subject of coryza.

Mouth.—In very many cases no lesions within this cavity are to be found. In other cases, however, flat shallow ulcers looking almost as if they were abrasions of the epithelial surface are to be found within the lips or on the gums. In the latter case they are sometimes found at the edge of the teeth. The lower lip is the more commonly affected. Careful observation shews that such ulceration probably has its origin in small round areas of thickened epithelium, which look at the first glance to be becoming vesicular, these become roughened on the surface and subsequently abraded. In some case where they are in close contiguity they merge giving rise in this way to irregular abraisons or ulcerations. They are seldom very vascular, but where in close connection with the teeth the condition is more irritable looking, probably owing to the action of the food in chewing.

In other cases and especially where the sickness is protracted, I have found the ulcerated condition most marked along, at the base of the gums and at the reflection towards the buccal surface. In such cases the ulceration extends in an almost unbroken line from the angle of one side to that of the other, and where such an extent is involved the abraded surface is angry and vascular.

The papillae at the inner angles of the mouth are also then involved; they are congested and covered with thickened epithelium, which, at parts abraded, leaves angry ulcerated surfaces.

Tongue.—In such cases as shew irritable conditions of the buccal mucous membrane, the tongue is sometimes also the seat of pathological change.

The surface shews some congestion, the epithelium looks in part sodden, thickened and more opaque than in health. In parts towards the posterior aspect irritable irregularly abraded patches may be seen. The mucous membrane of the under surface from the tip passing backward along the lower borders is commonly thickened, and in a few cases I have found distinct ulceration. The anterior portion of the upper surface is usually healthy, but in some cases small circular patches of thickened epithelium are to be observed. Ulceration is not common on the anterior surface, but if present may be looked for at the posterior portions of the dorsum and towards the lateral margins. The ulceration in this area is probably in part due to the pressure of the fauces and palate in deglutition as Dr. Sanderson has suggested.

Palate.—In most spontaneous cases dying without treatment the palate is nearly healthy. The simplest form of pathological alteration in this situation consists in pappular thickening of the epithelium, but where ulcerated conditions are well marked in the mouth, we are liable to find that such changes may also be found here. In the worst cases irregularly abraded ulcerated areas may extend over nearly the whole of the upper surface of the mouth, but in milder cases of ulceration these are restricted for the most part to the posterior portion.

For the most part such conditions are not well seen in spontaneous cases, as these most usually die within a fortnight of the date of infection, but, in cases where by the use of serum the disease is protracted, lesions in the mouth assume a very severe form and quite conform to the classical descriptions of Rinderpest as it has been found to occur in Europe.

Nose.—The mucous membrane generally and over the septum in particular is deeply congested. It appears of a purple colour and is covered over with tenacious mucus. Similar pappular thickenings of the epithelium may also sometimes be seen to those which occur on the mucous membrane of the mouth. I have not encountered any ulceration.

Eyes.—The conjunctivœ are always congested, and in most cases the early bright red appearance of the inner angles is characteristic.

Tonsils.—These are invariably enlarged and congested. In many cases when section is made small pin-head abscesses are found in the cortical layer. The pus found in this situation contains very frequently streptococci of a peculiar species, and as a similar species has been seen in and separated by me from the blood, I incline to the view that it had obtained access to the latter by this channel.

Sublingual Glands.—These are also always congested and edematous, but never the seat of abscess formation.

The glands surrounding the larynx are likewise enormously enlarged, congested and edematous.

Respiratory Tract.—The larynx is invariably congested, but in some cases only to a slight degree. In other cases the congestion is so great as to give it a purplish colour throughout. There is also in some cases evident signs of edema of the submucous tissue, and in a few cases small pin-point ecchymoses are to be seen between the arytenoid cartilages and the vocal cords.

All over, but principally in relation to the vocal cords, an exudation of mucus is to be found covering the epithelium.

Trachea.—More or less generally there is to be found congestion. In many cases it is so slightly marked as to be scarcely beyond the normal, while in others the condition is more decided. In some few cases I have found small submucous petechiæ.

Lungs.—The lung tissue is generally perfectly healthy, but in some cases irregular patches of collapse are to be found towards the free edges.

In many cases congestion is found, but I am of opinion that that it is almost invariably hypostatic.

The only peculiar feature which I have seen consists in the presence of interlobular emphysema. In many cases it is circumscribed within small limits of one lobe and is rarely diffuse. Where animals in the last stage of the disease are killed it is almost invariably small in amount; sometimes absent. Extensive patches are mostly found in more protracted cases, and I consider the cause to be almost purely mechanical, and the condition expressive of difficulty in the respiratory efforts occurring in the later period of the disease.

Circulatory System.—The pericardium in some rare cases contains a slight amount of fluid.

The parietal lining in some cases shews the presence of a few subserous hæmorrhages.

Heart.—On the outer surface it is common to find subserous extravasations, which in their simplest forms, consist of minute dark red points; where, however, many occur together, large irregular patches result at the outer borders of which discrete petechiæ are to be found extending for some distance around.

The commonest seat of such a condition is in or close to the inter-ventricular groove. Miscroscopic examination shows that the blood so extravasated is not limited by the surface of the muscle on the one hand and the serous covering on the other, but dips between the fibres of the muscle itself to some little distance.

The muscular tissue is usually paler than normal; the striation is more or less obscure, and the condition of cloudy swelling almost invariably is present.

Cavity of the Heart.—Coagulated blood is commonly found in the ventricles, and is usually in the form of soft clots. In some cases the blood has not undergone coagulation in a perfect form, but appears dark and thicker than arterial blood.

The endocardium usually presents numerous petechiæ, and the most common seat for these is in connection with the attachments of the "musculi pappilares."

Blood.—The blood is usually dark in colour, and, as a general rule, does not coagulate or only imperfectly so. I have frequently drawn blood during the latter stages of life into sterile vessels for the purpose of obtaining serum, but after forty-eight hours it has not been coagulated, or only in the form of a soft clot, which exuded a very small quantity of dark blood-stained serum.

Lymphatic Glands.—As an invariable rule the lymphatic glands throughout the whole body are enlarged and congested, frequently edematous. In some cases certain of the thoracic glands are little enlarged, but so deeply congested as to appear like blood clots; the others, however, are very much enlarged and edematous. The enlargement is best seen in the abdominal cavity, and especially in the mesentry supporting the small intestine.

Œsophagus.—In very many cases most careful examination is requisite to detect any departure from normal. Where marked congestion occurs in the pharynx it is common to find it extending some distance into this tube, but in cases where the mouth lesions are slight, the œsophagus may appear practically in a state of health. In some cases, however, slight pappular elevations of the mucosa may be seen, and in other cases small petechiæ are present. In other cases I have seen small bright-coloured irregularly spherical patches of a brilliant red colour, giving a similar appearance to flea-bites on the skin.

Rumen.—This viscus is usually found full of food, and I have not found in any case under my observation any evident pathological change.

Reticulum.—The contents of this stomach are usually normal, and the mucous membrane has been, in all cases I have seen, absolutely healthy.

Omasum.—The contents of this stomach are always very dry, indeed this condition is so marked that where the food is carefully removed in caked masses such pieces present a perfect cast of the mucus surface on which they have been in close adhesion. In some cases portions of the epithelial covering is removed adhering to the caked food material.

The only abnormality I have discovered consists in a slight general hyperæmia of the whole structure, but I have never observed any sloughing.

Abomasum.—In this organ, generally speaking, the most marked pathological changes are to be found, and while, in many cases, the alteration is of a pronounced type, and the destruction profound, in other cases, which have evinced all the symptoms of the disease and died, the changes are of the slightest.

The contents are generally more fluid than in health, and the odour is abominable. Indeed, the contents are commonly highly putrid. The changes produced here may be congestion, submucous extravasation of blood, thickening of the mucous membrane, abrasions, sloughs, and ulceration.

The congestion varies within wide limits; it may be of a dark rosy colour throughout, or may be a deep purple colour. It may be uniformly distributed, or may be in patches more or less circumscribed. Commonly, over the deeply congested areas will be found small petechiæ, which vary in size from that of a pin point to a pin head. About the pylorus it is common to find little elevations of the mucous membrane which usually surrounds the opening of a gland tube, and in some cases the orifice of this tube is filled with blood.

The abrasions usually arise, I think, from such thickened areas. Numerous small ulcers also are commonly present. These seem to take origin in connection with the tissue lying over the small extravasations of blood, and where several in close proximity coalesce, irregular ulcers of much larger size result. Sloughs are very common in relation to the mucous folds of the stomach, and in this situation have a tendency to be irregularly elongated in the direction of the folds. In most cases a definite edema is seen in the submucosa.

Intestine.—The conditions found in the higher part of the alimentary tract are here reproduced, but the congestion and submucous extravasations are commonly seen in a more violent form in the intestines. The disposition of congestion and ulcerations is irregular, so that it is a matter of difficulty to assign any peculiar liability of any part to special change.

Duodenum.—Is invariably congested, and this may be associated with submucous hæmorrhages and ulcerations.

The congestion and ulceration is commonly patchy, thus, while the upper third may be badly affected the lower may be nearly free from any inflammatory action, or the upper and lower thirds may be comparatively free while the middle may show broad patches two or three inches long, of violent inflammatory action.

Jejunum.—In some cases it is so slightly affected as to be almost normal, while in others it is the seat of the most violent inflammation, and may contain long cylindrical casts of the bowel. These casts are peculiar; the centre is often gelatinous-looking, with clots of blood mixed with it, while the outer surface seems to consist of exfoliated mucous membrane.

Illium.—This portion of the intestine may be similarly the seat of intense inflammatory action, with ulceration throughout, or may be simply congested with areas irregularly distributed of deeply congested or inflamed tissue with ulceration.

Around the ileo-coecal valve it is common to find deep congestions, and frequently submucous extravasations of blood.

Coecum.—This frequently is the seat of congestion merely, but in other cases it is violently inflamed; patches of ecchymoses are distributed throughout, and it is filled with blood-stained mucus.

Rectum.—The changes here are most varied; thus, while in some cases only a general congestion may be found, in other cases a violent inflammatory action may be found throughout the entire extent.

Peyers Patches.—On naked eye examination, in cases where the bowel lesions are severe, intense inflammatory action would appear to have invaded the Peyers patches. In the same case, however, one will find patches which are quite clear of inflammatory action, while others are apparently involved. Such a condition at once suggests that there is no lesion of such glandular structures essentially due to Rinderpest.

Microscopic examination fully confirms this, for in the worst cases one may find the mucous membrane entirely denuded over a patch, but the adenoid tissue or glandular structure proper is but little affected.

In such cases, as might naturally be expected, the gland tissue is congested and there is commonly present a very considerable amount of altered blood, which is found in the lacunæ of the adenoid tissue.

I do not therefore think that any specific change occurs here, and that at most these patches only partake of the general congestion which more or less obtains throughout the whole of the intestines.*

The Blood.—The blood as a rule is wanting in coagulative power, and is thinner than normal.

In the early stage of the fever there is a distinct increase of the leucocytes, and at a later stage a considerable number of poikilocytes are to be found among the red corpuscles.

At a later stage of the disease the small elements known as blood plates are less numerous than in healthy blood, but where the animal is recovering they are augmented in numbers.

As regards the cause of Rinderpest, I am not prepared to make any definite statement. The blood evidently contains innumerable infective particles in every drop, yet where blood is drawn from cattle in the early stage, that were formerly clean, and infected by subcutaneous inoculation of blood, our present methods fail to show any bacteria, or if they do these are only in cases where secondary infection cannot be eliminated. Further and prolonged research will be necessary to determine with any degree of certainty the true cause of this terrible malady.

APPENDIX 3.—EXPERIMENTAL INVESTIGATION.

RINDERPEST.

The earlier experiments which were made at Taungs in Bechuanaland, were more or less devoted to determining the most convenient and certain fluids which, taken from the bodies of animals suffering from Rinderpest, could be used as virus for the transmission of the disease to healthy animals.

Infecting Virus.—I finally decided that blood was a certain and convenient virus to be used for this purpose.

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* In one case I found great hyperplasia of the adenoid tissue, which had invaded the superjacent structures.

Method of taking Blood.—The methods by which blood can be kept for a time in a fluid state are at least two in number.

1st. By defibrination.

2nd. By means of neutral citrate of potash or soda.

The objection I have to the first method is that if the blood is defibrinated by whipping with a wire brush, there is the grave disadvantage that micro-organisms from the air find ingress and the use of such blood is liable to ensure the transmission of secondary infections. To overcome this one may have recourse to defibrination in a sterile bottle in which some glass beads have been previously introduced.

Citrate Method.—By using citrate of potash it is possible to draw off the blood aseptically, and I consider this method by far the best. The method is as follows: Bottles, each capable of holding about one litre of fluid, are cleaned, and the mouths are then closed by cotton wool plugs in a manner similar to the test tubes which are used to contain sterile media for bacteriological purposes. To each bottle is then added about 50 cubic centimetres of a five per cent. solution in water of neutral and chemically pure citrate of potash. I was formerly in the habit of using a greater quantity, but find by experience that it is not necessary to use much of the solution. After the citrate solution has been placed in the bottles and the cotton wool plugs replaced they are to be sterilised in the autoclave, after which they are ready for use. In the same way test tubes can be prepared and serve most usefully to collect blood with which it is intended to make inoculations into either animals or media.

To obtain blood the following procedure is adopted: A Robertson cannula (a long thin straight cannula with a very sharp incurved point), is sterilised by thorough boiling while contained within a long glass tube that is closed by rubber corks at each end. A piece of rubber tubing about a foot in length, carrying a short tapering glass nozzle is also sterilised in a suitable vessel. The animal, having been cast, is shaved clean along the site of the jugular vein. The skin is then washed with soap and water by well brushing and thereafter with water. A fresh washing is then given with hot carbolic lotion (strength 1 in 20). All knives, needles, gut, &c., are kept while not in use in the same lotion.

Operation of Bleeding.—In operating for the insertion of the cannula a short clean incision is made over and parallel to the jugular vein, and while the vein is made tense by pressure below the site of the incision the point of the cannula is made to enter the incision and then thrust into the vein. The blood will now issue freely from the nozzle and the flow can be arrested from time to time by making pressure on the tube. By steady dexterous movement one can remove a plug from a bottle or test tube and insert the glass nozzle, when, the pressure on the tube being relaxed, as much blood can be taken into it as is desired.

Citrate Method for the Preparation of Cover Glasses.—Where it is desired to make a microscopic examination of the blood the test tube containing the citrate solution should be emptied by inverting it after withdrawal of the plug, and although all the solution that can by this means be got rid of is removed, yet it will be found that the mere traces which remain will prevent the coagulation of the blood.

Incubation Period in Rinderpest.—By subcutaneous inoculation of healthy animals with one cubic centimetre of blood, taken as described, it was found that after a period, which varied from 54 hours to six days, a rise of temperature occurred, and within 24 to 36 hours later the thermometer would register 104 degrees to 105 degrees Fahr. The style of the temperature curve is markedly characteristic. The morning temperature is always, as a rule, lower than the preceding night but higher than the previous morning but just at the height of the fever it will be found that the morning temperature shews very little remission from that of the night before.

Inoculation by means of virulent blood, more especially if the blood has been taken from a former case also infected by the subcutaneous injection of the blood is more certainly fatal if left to run its full course than spontaneous infection. It has, however, to be noted that the virulence of spontaneous infection is subject to very considerable variation.

Duration of Disease after Blood Inoculation.—Animal No. 9, inoculated with 2 c.c. virulent blood (citrated), died on the eleventh day subsequent to inoculation. Of three animals (10, 11, and 12), inoculated with 2½ c.c. citrated blood from a different case to the preceding, No. 10 died on the 9th, No. 11 on the 10th, and No. 12 on the 11th day subsequent to inoculation.

Influence of Physical Change in the Blood.—The addition of glycerine, both in the pure state and diluted, with water in equal proportions, to the fresh unaltered blood which was made to mix with these fluids directly as it issued from the vein, seemed to effectively destroy the contagium inasmuch as when the clot which immediately formed was forced through fine muslin and used to inoculate cattle, in doses of two c.c., neither Rinderpest nor immunity was produced. See Nos. 1, 2, 3, 4, and 5 of Appendix Similar results were obtained when weak glycerine and water containing small quantities of sulphuric acid (1 in 1,600 and 1 in 3,200) were used to mix with fresh blood.

Where, however, citrated blood was used, mixed with glycerine in equal proportions to inoculate cattle they developed virulent Rinderpest. See Nos. 13 and 14 of Appendix.

I am therefore of opinion that coagulation has a destructive effect on the contagium but that the destruction is limited only to the material enveloped in the clot is shewn by allowing virulent blood to undergo spontaneous coagulation, and after pouring off the clear serum if the dark bloody fluid which surrounds the lower part of the clot is used within 48 hours to inoculate cattle, Rinderpest is usually, though not always, produced (Nos. 209, 181, and 181A).

Spontaneous Attenuation of the Virus in Blood.—Citrated blood when kept for nine days loses its virulence (at Taungs where these experiments were made the mean daily temperature was about 100 degrees Fahr. in the shade). Animals inoculated with 2 c.c. of such blood do not become affected with Rinderpest. See Nos. 1, 2, 7, and 8.

Production of Modified Disease by Blood Inoculation.—If such blood is used to inoculate healthy cattle on or about the sixth day after withdrawal from the body, a modified attack of the disease, with recovery, may ensue. (See No. 6.) During the past year I have been largely employed in rendering assistance to the Veterinary Department by furnishing them with virulent blood to be used for the purpose of infecting clean cattle that were destined to produce Rinderpest bile for inoculation purposes. As this blood had to travel over considerable distances by rail or cart, it is obvious that the employment of defibrinated blood had the grave disadvantage attaching to it that it would be putrid before it could be used, while by the employment of sterilised bottles and by bleeding the diseased animals aseptically into citrate solution we were enabled to send it satisfactorily. On one or two occasions, however, where, either by over-carriage on the railway or other delay, the blood was used at a late period, it either failed to produce the disease or did so in such a modified form that the animals recovered and were fully protected against subsequent spontaneous infection (where animals are protected from any special disease in South Africa they are spoken of as being "salted").

It has been asserted that this modification or attenuation of the virulence of the virus was due to the use of citrate, but facts at my disposal prove that equal results are obtained when defibrinated blood is used under equal conditions of keeping; thus, to quote an instance, defibrinated blood was sent from the bile station at Aberdeen to Jansenville and used to inoculate about sixty or more head, all of which took the disease so mildly that they entirely recovered. This has been the most notorious case of cattle being inoculated with the modified form of the disease in the whole period of Rinderpest inoculation in South Africa.

Virulence in Relation to Period of the Disease.—The virulence of the blood from an infected animal has a certain degree of variation in relation to the period of fever In my experience the blood, if taken from an animal during the second or third day of fever, and while the temperature is still rising, is most virulent, and I prefer to use such blood for purposes of fortifying the blood of "salted" cattle. If one bleeds an animal whose temperature is declining, and whose general appearance gives indication of probable recovery, and if such blood is made use of for inoculating healthy cattle, it is probable that the disease conferred will be modified in type and recovery ensue. I have had such an experience in two animals where the blood used was drawn into citrate solution and immediately used, but the same blood after having been kept for two days and being then used to inoculate two cattle produced virulent Rinderpest.

So far, therefore, as this experience goes, it would seem to indicate that the sojourn of the virus in the presence of a very small amount of citrate of potash permitted of the virulence of the virus becoming raised.

Influence of Dessication on the Virus.—With a view to determine the manner in which the infection of Rinderpest is spread I have made a considerable number of experiments with dried blood. Where citrated blood was dried in a fairly thick layer by artificial heat and subsequently ground to powder, dissolved in water, and then used for inoculation into clean cattle, no Rinderpest followed, even when a quantity so large as two grammes was used. (Nos. 62, 64 and 65). Where, however, the citrated blood was used immediately after being drawn, to smear over sheets of glass, which were then partially dried in the air of a warm room, and subsequently in the dry air incubator: inoculation with 2 grammes of such blood produced most virulent Rinderpest.

I also dried blood under conditions which might be considered as a near approach to what would occur in nature. To this end clean sawdust was dried and some citrated blood being mixed with it and rubbed up by hand, so as to make the sawdust bright red in colour and scarcely damp to the touch, it was dried at room temperature for six days and finished off in the incubator.

Two clean animals now received 2 grammes each by the mouth, which was simply opened, and the powder distributed inside.

The temperature of the one animal rose in the evening of the sixth day, while in the other it rose on the morning of the eigthth, both developing virulent Rinderpest. See Nos. 192 and 193.

Blood dried artificially in a thick film and afterwards reduced to powder was also given to a series of cattle, fourteen in number, one of which only developed Rinderpest, and hence it is not possible to definitely assert that the disease in this latter experiment had been produced by this means.

In order to determine a convenient method of preserving and using Rinderpest virus, and also whether by keeping the blood for varying periods before drying, it would not be possible to get an attenuated virus, a series of experiments were conducted in the following way:—

Different lots of knitting wool cut into short lengths were soaked in blood which had been kept during 24 hours, 51 hours, and 72 hours respectively. After drying in the air of a tent during an intensely warm day, one animal was inoculated with one cord each by means of a seton needle, the cord being drawn under the skin of the dewlap and there allowed to remain. No. 111 was inoculated with a cord (24 hours' kept blood) six days later with a fresh cord, on which blood had been dried after only two hours' keeping. The temperature rose six days later, and Rinderpest was produced. No. 109 had a 51-hour cord, and No. 110 a 72-hour cord. In the case of the latter two no rise having occurred during fourteen days, each was inoculated with a 2-hour cord. In the case of No. 109 the temperature began to rise on the twelfth day, and in the case of No. 110, on the seventh day. It is, of course, to be noted that the woollen threads carried only a very small amount of blood.

No. 165 was inoculated with three double cords carrying blood in which they had been soaked immediately after the blood had been obtained, and subsequently dried. The temperature in this case rose on the evening of the fourth day, and Rinderpest was produced. In my experience I have found that the virulence was best conserved in blood which was as quickly as possible dried with free access of dry air and at as low a temperature as possible compatible with successful drying. Its virulence is not interfered with if subsequently placed in a hot incubator, provided that no traces of moisture are present.

Disinfection of Hides.—These experiments are of extreme importance from the point of view of the disinfection of hides. For such materials strong carbolic washes cannot be used without the risk of serious damage to the articles exposed to it, but it is evident that if the articles in question are thoroughly soaked either with water only or in a weak antiseptic solution and subsequently dried in a hot sun, that complete disinfection may be attained. In cases where such hides are to be exported, it would be most advisable as a complete check and safeguard to have a final dipping at the port of shipment or at the port of discharge.

Spread of Rinderpest by means of Dried Virus.—I am confident that Rinderpest is mostly spread from place to place by dried virus, and as my experiments show that where the virus is quickly dried in a thin layer, the virulence is better conserved, it is obvious that hands, boots, and garments which come in contact with infective material are to be regarded as dangerous agents in the spreading of the malady.

The Nasal Mucus as an Infecting Virus.—In case of spontaneous infection the discharge of nasal mucus from the nose is a characteristic symptom.

I have experimented on very numerous occasions with this material, principally with the object of determining its infectivity. If one receives some of this mucus, from the nose of a *spontaneous* case of Rinderpest or from a case infected in the manner I am about to describe, at or about the third day of fever, and if one uses the sponge wetted with the secretion to rub on the nose and the inner part of the upper lip of a healthy animal, the disease certainly follows.

Period of Incubation after Infection with Mucus.—If the mucus is thus transferred directly a rise of temperature will follow within six days; the shortest incubation period I have recorded is 51 hours, and I believe that where the infecting is carried out in this way the incubation period is of the shortest. Infection may be made in the same manner by using virulent blood, but the incubation period is relatively longer when this material is used.

Effect of the Nasal Mucus which has been kept.—Where the nasal mucus is not at once used but kept for a period of 24 hours, exposed more or less to air, infection is not produced with the same certainty, and in some cases either failure or a modified attack may result.

In the case of No. 35, infected at Taungs with mucus brought from Phokwani and kept for 24 hours, a modified attack was produced which went on to full recovery, the animal only being slightly ill during this period. See No. 35.

Rinderpest Virus is readily Attenuated but cannot be Maintained in such Condition.—From the experiments already referred to it is seen that the virus of Rinderpest readily attenuates, and if the condition of attenuation of the virus which is produced about the sixth day (under conditions of temperature already mentioned) could be maintained for a time, especially if the time were long enough in duration to admit of the degree of attenuation being tested, it is obvious that a method of inoculating with attenuated virus would be in our hands, of a character nearly approaching the ideal, Unfortunately, however, it has been shown that the process of attenuation does not arrest itself, nor is any method now known by which the arrest can be accomplished, and hence within a very short period the virus passes spontaneously from a state of virulence through a stage of attenuation to that of absolute inertness.

Foiled in the attempt to arrive at an attenuated virus in this way, I was subsequently led to direct my attention to the condition of the virus in a dried state, and as indicated I found that when blood in which coagulation had been arrested was dried in a thin layer quickly that it produced virulent Rinderpest, and since blood which had been kept for a time and dried in a thick layer did not produce the disease, I had hopes of determining a method under the operation of which one could dry blood in such a fashion that virulence in an attenuated form would be produced. Numerous and carefully-planned experiments, however, have shewn me that it is impracticable to obtain an attenuated virus with any degree of certainty by such a method. Had such a method succeeded it is obvious that it would have been of the utmost advantage, because large quantities being made at one time it would have been easy to have tested a quantity and thus ensured that the whole batch would answer the desired end when used in practice by the farmer.

Production of Weak, Passive Immunity.—During the period in which I was experimenting with blood which had been kept for a period and thus lost virulence, it was remarked that if the dose was anything greater than 2 cubic centimetres that a slight rise of temperature was commonly seen about the third or fourth day.

This peculiarity (Nos. 5, 7, 60, 61 and 54) perplexed me, and indeed for a time led me astray, as, occurring as it did, at a period which agreed with that of the first rise of temperature after the inoculation of virulent blood, I was led to consider it as possible that the virus in the blood used was not quite inert, and that it was giving rise to abortive attacks of Rinderpest.

Toxic principle Present in Blood.—As, however, the same thing occurred when inert dried blood was dissolved in water and injected, I began to suspect it as being a reaction due to the presence of some toxic principle in the materials used. To determine this, some blood was taken in citrate solution in a long narrow bottle and permitted to stand for some days. The action of the citrate, in preventing coagulation, allowed of the corpuscles falling to the bottom and leaving clear plasma above. This was carefully drawn off and passed through a Pasteur filter, so that the fluid passing through was absolutely free from corpuscles and micro-organisms, and was used to inject into three animals, one of which received 30 c.c., one 20 c.c., and one 10 c.c. In each case there occurred a transcient rise about the fourth day, and I felt satisfied that the blood and plasma of Rinderpest animals, while being unable to produce Rinderpest, owing to the living virus being dead or removed, contained a toxic principle which had some action, though slight in degree, when inoculated into a healthy animal, and of which the transcient rise of the animal's temperature was evidence. Of two animals which had been twice inoculated with glycerinated blood early in the course of my investigations, and subsequently inoculated by smearing their noses with virulent mucus one died while the other recovered, and this fact, in the light of later experiments, led me to experiment somewhat largely with fluid and dried blood which had lost its power of inducing active Rinderpest.

Immunising action of Glycerinated Rinderpest Blood.—The material used for the purpose of inducing passive immunity consisted, in the first instance, of citrated blood, which, after a longer or shorter period of time, during which it had been kept free from putrefactive change, was mixed with glycerine and kept for a number of days longer. The object of adding glycerine was mainly for the purpose of preserving the material from putrefactive change, and allowing at the same time the virus to become inactive. At first I experimented by making use of a number of samples which had been kept for varying periods of time previous to the addition of glycerine, and where I refer to any sample as an eight day, six day, four day or two day, it is to be understood that this expresses the period during which the blood had been drawn from the body of the sick animal, and on the completion of which period glycerine was added. The first experiment was made on four animals, Nos.

48, 49, 50 and 53; No. 51 being kept as control. The four animals were each inoculated with 10 c.c. eight day blood, three days later with 10 c.c. six day blood, three days later with 5 c.c. two day, at which moment the control was also inoculated with 5 c.c. of two day blood.

Six days after the first inoculation No. 48 was found to have an elevated temperature, and later was found to be suffering from Rinderpest in a mild form. No. 49 was inoculated with 2·5 c.c. virulent blood fourteen days subsequent to the last inoculation, as a result of which it developed Rinderpest and died. Nos. 50 and 53 were each inoculated with 2·5 c.c. virulent blood four days after the last inoculation with glycerinated blood, both took the disease in a modified form, one dying, while the other (No. 50) was already recovered, within twelve days from the moment of virulent blood inoculation.

In another experiment, Nos. 69, 70 and 71 were each inoculated at one time with 30 c.c. of two day glycerinated blood. Five days later No. 69 was inoculated with 2 c.c. virulent blood, while six days later Nos. 70 and 71 were each inoculated with the same amount of virulent blood. In all three cases the disease was produced, but so modified in character that No. 69 had absolutely no symptoms whatever except temperature. In the case of Nos. 70 and 71, however, while the disease was ushered in in a mild form, it gradually became more severe, and both animals died.

In the case of Nos. 55, 56 and 57 the animals were each inoculated with filtered plasma obtained by allowing the corpuscular matter in citrated blood to subside, drawing off the plasma and filtering it through a Pasteur filter. Of this filtered plasma No. 55 received 30 c.c.; No. 56, 20 c.c.; and No. 57, 10 c.c. Six days later they were each inoculated with 2·5 c.c. of virulent blood, as a result of which all three took Rinderpest: two died and one recovered.

Passive Immunity from Injection of Dried Blood Dissolved in Water.—Experiments were also made with dried blood which had been dried under such circumstances as ensured a destruction of the virulence. Such blood was dissolved in water and then inoculated, after which at varying periods inoculation of virulent blood was made and results similar to the foregoing were obtained. These results may be summarised by saying that where animals were first inoculated with inactive Rinderpest blood, either glycerinated or dried, that some peculiar change had been produced within their bodies, because when subsequently inoculated with such enormous doses of virulent blood as 2·5 cubic centimetres, they did not all die, and in all the produced disease was mild in character.

Effect of Injection of small doses of virulent Blood after Production of weak Passive Immunity.—It was obvious, however, that the doses of virulent blood which I was giving were much too large, and accordingly I reduced them to 0·5 c.c. in the first instance, and still later to 0·2 c.c. I eventually found that where inoculation was made with 30 c.c. glycerinated blood by subcutaneous injection, and where the material was satisfactorily absorbed without a swelling remaining after a period of ten days, the effect was such as to give such passive immunity to the animals as enabled most of them to resist a dose of 0·2 c.c. of virulent blood.

Disadvantages in the Use of Glycerinated Blood.—There are, however, several disadvantages in the using of glycerinated blood as a method of obtaining passive immunity. These are: firstly, the difficulty which would exist in drawing blood aseptically in the field; and secondly, the tendency, owing to the amount of corpuscular matter contained in it, for it to cause a swelling that is not very quickly absorbed.

Disadvantages Against Use of Dried Blood.—Against the use of dried blood is the objection that it is not readily dissolved, and the possibility that in practice, owing to the objection mentioned, the full dose might not be given.

Dr. Koch's Discovery of Immunising Property of Bile.—While I was experimenting with blood, Dr. Koch had made the important discovery that the bile, when taken with the precautions and under the conditions formulated by him, produces immunity against Rinderpest.

The conditions, among others, involved the use of dark green bile having a white froth and no putrefactive odour. Compliance with this necessitated that a great number of biles would require to be discarded owing to the fact of their colour being otherwise than that specified or objectionable as regards odour.

Glycerinated Bile.—It therefore seemed to be that the peculiar body which was present in the blood would also be present in the bile, and that if the bile were treated in the same manner in which I had treated blood, namely by adding glycerine and subsequently keeping it for a period before using it, that probably equal or perhaps better results might be obtained.

In the first instance the biles with which I experimented were such as departed but little from the characteristics of those as described by Dr. Koch. They were invariably taken after death. Out of three taken one was a heavy green bile with a tinge of brilliant yellow, while two others were more or less green with a brownish or yellowish brown tinge. In taking bile and preparing it, the following procedure was at that time adopted. I tied the neck of the bladder with two strong ligatures of grey twine, and cutting between these I removed the gall bladder intact which, after being immersed in a bucket of disinfectant and then washed in water, was opened over a clean beaker by a small sharp-pointed knife. When the bile was satisfactory, I measured it carefully and added to it half its volume of glycerine, so that the resulting mixture contained two parts bile and one part glycerine, various proportions were tried but this had my preference. After keeping the mixture for eight days, so as to ensure the disappearance of all active infective power, it was ready for use. In practice I inoculated at first 15 cubic centimetres into the dewlap of a mixture in which the bile departed but little from Dr. Koch's standard. When after later experimentation I found it possible to use other biles, and when moreover I had found *that some green biles taken from Rinderpest animals had little or no immunising power*, I determined upon mixing many biles together, and after admixture with glycerine, using 20 cubic centimetres to inject into animals of average size. I thus allowed for the possibility of some biles having little immunising power by the mixing of numerous biles, and the increased dosage used.

Experiments with Glycerinated Biles.—With regard to glycerinated green biles taken after death at full time of disease, I found that if an average-sized animal was inoculated with 15 cubic centimetres, and the part gently rubbed after injection so as to aid dispersion, that a subsequent injection of 0·2 c. c. of virulent blood mixed with water did not produce active Rinderpest. I may here refer to several experiments where numbers of animals were inoculated.

(1). Ten head of milk cattle sent up by train from a clean area in the Colony to Taungs. Each of these were inoculated with 15 c.c. of glycerinated bile by Mr. Duncan Campbell, M.R.C.V.S., Assistant to the Colonial Veterinary Surgeon. They were more or less exposed to infection on the same day, being mixed with six cattle that were suffering from Rinderpest. My staff and self left Taungs on that day, and the cattle in question were left in charge of herds. At a later date Mr. Robertson, M.R.C.V.S., prcceeded to Taungs from Kimberley and inoculated them with 0·5 c.c. of defibrinated virulent blood, which Dr. Turner kindly gave me from the Victoria Compound. Of these cattle one died and the others which were also visibly sick, made a good recovery.

(2.) Twenty head at Kimberley. These were inoculated with a mixture of glycerinated blood and glycerinated bile, and received 0·2 c.c. of virulent blood. None of this herd died, and though kept running with infected cattle, were all alive three months later.

(3.) Three head of cattle at Kimberley. Two of these were each inoculated with 15 c.c. of glycerinated bile, while the third received 12 c.c. of glycerinated blood and 8 c.c. of glycerinated bile. Ten days later they each received 0·2 c.c. defibrinated virulent blood. None died. Eight days later one of the first and the last each received 2 c.c. of virulent defibrinated blood without any evil effect.

(4). Eleven animals at Kimberley.

The first four pairs and the last three single animals in the series were each inoculated with different biles, of which only two could be said to be good green biles; two were yellowish green, one brown, one brown red, and two very pale yellow. One of the singles which was inoculated with a pale yellow bile that had only been kept 24 hours in glycerine suffered from a rise of temperature three days later, which proved to have been the onset of Rinderpest from which he died. None of the others suffered, although they each received 0·2 c.c. of virulent blood which had been taken by the citrate method and whose virulence was controlled and proved by inoculation into a clean animal.

(5). The Belmont experiment.

At the request of the Colonial Veterinary Surgeon I gladly acceded to the making of a large field experiment, asking him to kindly select the area for experiment and the herd, and to put the experiment in the hands of one of his assistants. Mr. Borthwick, the senior assistant in the Veterinary Department, was accordingly instructed to select the herd, which he did at Belmont.

It was intended that Dr. Turner should also go to the same herd and inoculate a portion of them on the Koch system, but I understood that want of sufficient bile prevented his being able to undertake his experiment at the same time as mine. On proceeding to the farm selected, belonging to Mr. de Villiers, with Messrs. Robertson, Borthwick, and Dudgeon, a herd of 128 animals was set apart for my experiment. This area was understood to be clean, but we found to our great dismay that the adjoining farm was already infected. After taking temperatures, however, I considered that there was no evidence of active disease, and I determined to go on with the experiment. One hundred and twenty-six animals were accordingly inoculated with glycerinated bile, and it was intended to give them 20 c.c. excepting the smaller animals which should have less. In the hurry of inoculating, two syringefuls each were given to the ordinary sized animals, but I found when I later examined the syringes that they each held rather more than 10 c.c. each, hence many had more than 20 c.c. each, while the smaller had about 16 c.c. each. Two animals were left as controls for the virulence of the blood which would be used ten days later.

On my return, ten days later, from Steynsburg, with an additional Veterinary Surgeon, Mr. Freer, M.R.C.V.S., I met Messrs. Borthwick, M.R.C.V.S., Robertson, M.R.C.V.S., and Mr. Dudgeon with Dr. Kolle (Dr. Koch's private assistant).

On proceeding to the farm we found that one animal of the 126 was already dying from Rinderpest, having shewn suspicious symptoms soon after our former visit and thus proving his infection to have taken place before the period of bile inoculation, and I think undoubtedly contracted from the adjoining farm which was only separated by a wire fence. Of the 125 animals remaining that had been bile inoculated, Dr. Kolle inoculated 10 with 0·2 c.c. virulent defibrinated blood which he had selected at and brought from the Victoria Compound at Kimberley, the remaining 115 and the controls were then inoculated with 0·2 c.c. of virulent blood, selected from and brought from the same place but from a different animal by Messrs. Borthwick and Robertson, M.R.C.V.S.

As a result of these later inoculations, the two control animals (inoculated only with the blood) contracted and died of Rinderpest after full period of sickness and within twenty minutes of each other. Two of the 115 were sick and recovered, none of the others were visibly affected, and although some calves which were born later took Rinderpest and died among the herd, all of these animals were in good health three months afterwards, at which period I received the last report.

After leaving Kimberley I proceeded to form a temporary camp at Steynsburg for the purpose of forming a bile station and to supply virulent blood for the starting of new Rinderpest bile stations in surrounding areas. The farm selected was that of Mr. Pretorius, and as he had running there about sixty cattle I was desired to inoculate them. This was accordingly done, and of these seven died, all the rest being more or less sick and eventually recovering.

It was worthy of note that in this case, when the inoculations were being performed, the cattle were thrown and held by the same Kafirs who were employed to work among the infected cattle on the bile station. Furthermore, five animals were particularly wild and were held by the noses by the hands of the natives, and these five were among the number that died. All the remainder of the herd were subsequently running loose on the bile station, being daily in contact with animals that had been infected either with blood or from contact with sick animals, and, according to a recent report, all are alive and well.

In the same district, infection having already appeared in two clean herds, Mr. J. A. J. Vermaak, a noted breeder of fine pure Friesland stock, placed his whole stock at my disposal for experiment at his own risk. In all, this gentlemen had (including about forty of his native stock), 341 head of cattle. I inoculated all these, first with glycerinated bile, and subsequently with blood. Out of these nine animals died. All then seemed to go well until the 23rd day after inoculation, when several appeared sick and (including calves), six died. This infection occurring after the inoculation was over, was extremely disappointing, as one could not be sure that the sickness would not spread through the whole herd. I was much relieved to find it come to a standstill, and in my record of subsequent experiment will be found the explanation that some animals are more readily immunised than others, and that the immunity acquired is in some cases (even occasionally among salted cattle) inclined to break down.

In connection with this form of inoculation several very interesting facts have been elicited. After the inoculation with glycerinated bile, as a rule no rise of temperature occurs if the bile is quickly absorbed. If it is merely injected under the skin into the loose tissue there is a decided tendency for it to set up irritation and to become encysted. Where this occurs it has been proved by experiment in my hands that very little immunity is established, and the swelling eventually forms itself into an abscess. Using equal doses of the same bile I inoculated ten head in which the injection was made into the dewlap and left there. In another series of ten the bile, after injection, was made to disperse by gentle rubbing. In the first series large swellings formed which eventually formed abscesses, while in the second the slight swellings which formed had almost entirely disappeared after three days. Ten days after the bile injection each animal was inoculated with 0·2 c.c. of virulent blood. All the first series became ill, and four eventually died. In the second series of ten animals no deaths occurred, and, indeed, excepting for some slight elevations of temperature, no illness was occasioned. This experiment was very remarkable, because in the first series young cattle were used which are more readily immunised, while in the second heavier animals were used. It is, however, to be noted that the experiments were made at different dates, and the blood used for inoculation was obtained from different animals. Admitting, however, the possibility of fallacy from the fact of the bloods having differed, it is worthy of note that the bad results obtained in the first series have been the worst results obtained in any of our glycerinated bile experiments.

In many cases following upon inoculation with virulent blood, animals previously inoculated with glycerinated bile shew no visible signs of illness at all, yet in very many instances, should there be cows in calf among them, it is common to find the cows aborting. Curiously it has been found that the aborted calves are in some cases born while suffering from Rinderpest, and in other cases the dead calves have shown intestinal lesions of a pronounced type such as are characteristic of the disease.

Duration and Character of Immunity following Inoculation with Glycerinated Bile and subsequently a Small Dose of Virulent Blood.—Experiments which have consisted in inoculating such animals with a second and larger dose of virulent blood at a later date, have shewn that where an animal developes a slight rise of temperature from the first blood inoculation it is more highly immunised than those in which the temperature has not risen.

Among inoculated herds if any sicken and die as a result of the blood inoculation, it is found that the deaths begin about the eleventh day and continue till about the sixteenth.

In some cases it has been found that deaths begin again to occur in the herd about the twenty-first day, but as a rule only a few die and the herd becomes clean.

The explanation I have to offer for this second occurrence of deaths is that the animals have become infected by the mouth before the period of immunity had been arrived at. Experiments have shewn, even where one produces a mild attack of Rinderpest by inoculation with blood that has been kept for some days, and where recovery supervenes, that if, when the normal temperature has just been regained, one infects the animals nose with virulent nasal mucus a sharp attack of Rinderpest follows. I conclude from such experiments that immunity is not immediately complete. The corollary to this argument is that relapses are due either to re-infection or auto-infection.

As further evidence that immunity is better established about a month after infection than at an earlier date, I might cite the fact evinced from numerous inoculated herds that where these are clean at such a period, it is rare to find infection occurring even when calves, born after inoculation, acquire the disease and die among the others. I consider, therefore, that the immunity which follows glycerinated bile and blood inoculations is at its best about two months after the date of inoculation. So far as our experience goes we have evidence to shew that at or about the completion of three months after inoculation we may expect to find infection occurring among some animals in herds that have been previously inoculated. The progress, however, of the disease under such circumstances is very irregular, as only one or two are affected at a time, and in them the disease is so very mild in character that they are readily cured by the use of serum, while on the other hand many recover without any treatment whatever if they are kept from drinking too much water.

The effect of blood inoculation is not equal in all animals in a herd although these have been inoculated with the same bile and afterwards with the same blood. In herds running in the open, where the temperatures are not taken daily at regular periods, one can only judge from what can be visibly seen, and my experience of such herds admits of my saying that, while no visible changes may be seen in some cases, in other cases some will sicken and of these a few may die. Where it is possible, as at this Institute, to make careful temperature observations, we have been able to determine that among cattle which shew no visible signs of sickness many may have a transient rise of temperature at or about the time when in an uninoculated animal we should find the onset of the fever occurring.

This rise of temperature, where it occurs, is most important from the point of view of subsequent immunity, as my experience goes to shew that such temperature reaction is followed by greater immunity than in the case of those which do not sustain the reaction. In other words, where the protection given by the bile is so great as to ensure no temperature reaction after the blood injection, the effect of the blood is of the slightest kind, but on the other hand, where either by the bile being weaker, the blood more virulent, or a special susceptibility on the part of the animal, the blood injection is followed by a definite rise of temperature, the effect produced is similar to what might be expected to follow a very mild attack of Rinderpest.

As I have said, generally speaking, we may expect the immunity from glycerinated bile, followed by blood, to break down after three months, but there is no certainty that it does so, as may be seen by the following experiment.

I purchased, in Grahamstown, two young cattle which had been inoculated along with the cattle of the town three months ago. Neither of these animals had been at all sick as the result of such inoculation, and I had the impression that a large enough dose of virulent blood would upset the remaining immunity, and admit of the Rinderpest being developed. To this end Mr. Robertson inoculated one with 50 cubic centimetres of virulent blood, but during the fourteen days which followed only a slight rise of temperature had occurred on two successive days To the other animal 100 cubic centimetres was then given, but no effect was produced. This result was most gratifying, and, judged in the light of experiments made, whereby a second blood inoculation was made very shortly after the first. shows most conclusively that the immunity from a single blood inoculation after bile is greater from two to three months after the date of inoculation than it is fourteen days after such inoculation.

In some districts animals among herds which had been inoculated, in which some had died, and the disease then disappeared, began to sicken about the twenty-first day after blood inoculation. Now we know that this period is one of danger should spontaneous infection get into the herd, but at that time we were under the impression that it was owing to immunity breaking down, or to the blood having failed to set up reaction. In view of the latter contingency I was led to advise a second blood inoculation of a whole centimetre of pure virulent blood eight to sixteen days subsequent to the first one. This has been very largely adopted throughout the Eastern Province and in some parts of the Free

State, and from the experience we have now gained it may be stated, as a general rule, that in herds where some mortality followed the first blood inoculation, a second blood injection was not attended with mortality, but in cases where no mortality or illness occurred as a sequel to the first inoculation it was common to have some sickness and mortality following the second. Generally speaking, the results have been very good, and as a means of protection I am inclined to consider the use of a second or third blood inoculation as well calculated to give a high degree of permanent immunity,

With regard to the results generally which have attended glycerinated bile inoculation followed by blood, I cannot yet speak fully, as the official reports are not all yet compiled, but the following reports convey information with regard to facts which it is impossible to overlook.

(i.) The Rinderpest Committee, Peddie, under date November 27th (several months after inoculation) states :—

> "I may inform you that the glycerinated bile method has been a thorough success so far. The district generally is highly satisfied, and many are repeating the bile inoculation."

(ii.) A report from Cathcart Station, dated November 9th, 1897, states :—

> "Number of cattle in district, 22,000. All these were inoculated with Edington's glycerinated bile between 15th and 24th August.
>
> All were subsequently inoculated with 0·1 c.c. virulent Rinderpest blood from nine to ten days following the bile. Some 4,000 were further inoculated with 0·2 c.c. virulent Rinderpest blood from 12 to 14 days following the first blood inoculation. On some farms this second blood inoculation caused a loss of about 5 o/o, and in two instances the percentage was raised to 40 and 50 o/o, but these were in small herds. Many animals which were treated with this second dose of blood sickened, and I am fully of opinion that the herds so treated have now a high percentage of salted cattle among them.
>
> However, the great majority of animals treated with the blood only once did not visibly react, so that would lead to the conclusion that they are not permanently immune according to the generally accepted belief.

Outbreaks have since occurred, and are doing so still, but the disease seems to be confined to one or a few animals, and at intervals. These have been treated with serum obtained from Kimberley, sometimes successfully, and at other times it has been a failure.

The difficulty met with is that farmers find it impracticable to go over their herds taking temperatures, consequently it is left till animals show visible signs, and in my limited experience serum has not proved a panacea in such cases.

At the present moment there is very little sickness in the district, but as three months have elapsed since the bile inoculation, we may now look out for the disease shewing itself.

I have been unable to account for more than 400 animals which have died from Rinderpest. This would make the percentage under two, and these losses have not been among bile inoculated animals in the main, but amongst herds which were infected and treated with 30 c.c. of defibrinated immunizing blood obtained from Kimberley.

Certainly Edington's bile has proved of immense value in this district, and in my humble opinion the mixing with glycerine does not seem to have interfered with the toxic or anti-toxic value of the bile, as our results, I think, distinctly prove."

(Signed) JAMES FORD,
Veterinary Assistant in Charge of
Station.

(iii.) At a still later date [28th December, 1897,] the following resolution of the Rinderpest Committee of the same district was received by me from the Chairman, the Resident Magistrate of Cathcart:—

Resolution passed at a meeting of this Committee on the 22nd instant. "This Committee begs to place on record the entire success of inoculation of the cattle in the Cathcart District by Dr. Edington's method, and would convey to Dr. Edington its entire satisfaction of results which were carried out four-and-a-half months ago. This now places us within reach of further scientific research."

(iv.) Veterinary Surgeon Armstrong, M.R.C.V.S., writing from Graaff-Reinet, states as follows:—

"Rinderpest is now appearing in a few herds of cattle inoculated according to your method three months ago, just one at a time becoming affected, and I find these cattle that have been inoculated respond more readily to defibrinated blood or serum than those which have not been inoculated with bile. We have some fortified salted cattle here, so we produce our own blood or serum.

> "Out of 10,000 cattle inoculated by your method here, nearly all two blood inoculations, official returns showed a loss of 44, but we only used the best bile, and had no surplus from 5 per cent. sent in."
>
> (Signed) H. J. ARMSTRONG.

While records such as these may be shewn for successful results, it has to be admitted that in some districts considerable mortality has followed the use of this method, especially in the Koonap district of Fort Beaufort. At the same time, however, it deserves to be noted that almost coincident with the commencement of inoculation in Beaufort, Rinderpest had appeared, and was spreading rapidly on the commonage, while the adjoining district of Alice was already badly infected before inoculation could be begun. Apart, however, from the onset of spontaneous infection, I have to note that in one district where mortality was high a very great proportion of the animals dying had visible signs of Red-water or Texas Fever, and in this connection I would direct attention to the short statement regarding Red-water which appears elsewhere in this report.

It is a remarkable fact that all cattle are not equally easily immunised. Again, although cattle may withstand a certain dose of blood after bile inoculation, it is certain that a definitely increased dose can and may overturn the immunity, and admit of the onset of Rinderpest in full virulence.

Of 20 oxen which I had begun to immunize, all received after bile first 0·1 c.c., and then by periodic injection the following:—1·0 c.c., 10 c.c., 50 c.c., 100 c.c., 200 c.c., 400 c.c., 500 c.c., 1,000 c.c., 2,000 c.c. Up to the time of giving 20 c.c. of blood the following results were noted:—

No. 1.—No reaction.

No. 2.—Slight reaction up to 103 four days after first blood; on fifth and sixth days after 10 c.c. reaction of 105.

No. 3.—No reaction.

No. 4.—No reaction.

No. 5.—No reaction.

No. 6.—No reaction.

No. 7.—No reaction.

No. 8.—No reaction.

No. 9.—No reaction.

No. 10.—No reaction.

No. 11.—Slight reaction up to 103·4 four days after blood.

No. 12.—No reaction.

No. 13.—No reaction.

No. 14.—Slight reaction up to 103 four days after blood.

No. 15.—Slight reaction up to 103·3 fifth day after blood.

No. 16.—No reaction.

No. 17.—No reaction until fifth day after giving 10 c.c. when temperature rose to 103·2 then fell, next evening rose to 105·2, next evening to 106·4, next mid-day the temperature was 106·3 from whence it fell till death on the following day.

No. 18.—No reaction.

No. 19.—No reaction.

No. 20.—No reaction.

In this set of experiments the cattle were twice inoculated with glycerinated bile, and it is interesting to note that it is possible to raise the immunity up to the highest degree of fortification without much risk, for although in this case one animal died, it was not clear at the *post-mortem* examination that Rinderpest had been the actual cause of death, although I have no doubt that inoculation acting in an animal in which acute liver disease was present, it contributed to the fatal issue.

Inoculation of Glycerinated Blood and Virulent Blood simultaneously.—Having regard to the fact that one has to wait for the completion of a period of ten days subsequent to the bile inoculation before one can inoculate with blood, I made an experiment to determine whether it would not be possible to mix a definite amount of virulent Rinderpest blood with glycerinated blood, and use the mixture for inoculation. Accordingly, three animals, Nos. 119, 120, and 121, were each inoculated with 20 c.c. glycerinated blood, to which virulent Rinderpest blood was added in the following proportions, viz. :—

No. 121 had 20 c.c. glycerinated blood, and 0·1 c.c. virulent defibrinated blood,

No. 120 had 20 c.c. glycerinated blood and 0·2 c.c. virulent defibrinated blood,

No. 119 had 20 c.c. glycerinated blood and 0·3 c.c. virulent defibrinated blood.

As a result it was found that the temperature of the one having the largest quantity of virulent blood rose first, and the others in sequence proportionate to the amounts of virulent blood injected. All three developed modified Rinderpest, and while the first and second died, the third—that one having the smallest dose—made a full recovery.

Intravenous injection of Glycerinated Bile in an Animal already Sick with Rinderpest.—No. 162 was inoculated with 4 c.c. of pure Rinderpest blood. About 50 hours later the temperature began to rise, and after it had been elevated for four days, 20 cubic centimetres of glycerinated bile was injected into the jugular vein, and the same procedure repeated on the following day. The animal made a good recovery.

Since the date of the experiment it has been repeated on different occasions in the field, always with good results. In one case where infection had appeared in a herd this method was used throughout, and the disease was so thoroughly averted, the Colonial Veterinary Surgeon has informed me, that the owner of the herd would not believe that it had actually been an outbreak of Rinderpest.

It is also well-known among the Veterinary staff that in the case of herds which had been infected previous to inoculation in the ordinary manner with glycerinated bile, it has on a number of occasions followed that the mortality became arrested.

Dr. Koch's discovery of the Possibility of Immunising cattle against Rinderpest by means of Rinderpest Bile.—This discovery was the first real advance that was made in this country to check Rinderpest by means of inoculation. No doubt Dr. Koch had already shewn that the serum of salted cattle possesses protective power, a fact which has already been determined by Nencki, Sieber and Wyznikiewiez, and which, judging by the work carried out by Behring and others, was to be expected; but the discovery could not help us much for the simple reason that we had not salted cattle from which to obtain such serum. In justice to my staff and myself I may at this juncture state that early in the history of our researches at Taungs we took into consideration the question of beginning work with serum. Owing, however, to the fact that the situation of the camp was far removed from any town, that the temperature (over 100° Fahr. in the shade) was opposed to any satisfactory work with serum, and that no quantity of salted cattle was available, I was forced to abandon the idea, the more so as we believed we were on the track of a satisfactory method of dealing with the disease on other lines.

Dr. Koch's bile method was, however, a most practical and valuable discovery, and despite the fact of its having certain grave disadvantages, has proved of incalculable benefit in this country. To it in the first instance is to be ascribed the fact that we were able to "salt" large numbers of cattle, the acquirement of which has since admitted of work being taken up on other lines.

Dr. Koch discovered that if the fresh gall from an animal which had succumbed to the pest was taken, and of this 10 cubic centimetres was injected subcutaneously into a clean animal, that it became so highly immunized that 40 c.c. of virulent blood could be injected four weeks afterwards without evil effect. He found that this immunity sets in at latest on the tenth day, and that smaller doses gave uncertain results. He emphasized the fact that bile taken too early in the course of the disease did not immunize, and that bile taken from an animal which had died of sequelœ also did not immunize or only imperfectly.

In the directions given it is stated that the animal should be killed on the sixth or seventh day after the first rise of temperature or the fourth or fifth day of visible symptoms. To be useful the bile should be of a dark grass green, sweet-smelling and clear, without sediment.

Unfortunately, owing to the results which were obtained on inoculation in the field, that Rinderpest made its appearance in some herds, that numbers of cattle were simultaneously affected, and that the time which had elapsed between the moment of inoculation and illness agreed with the period of incubation, it became evident that in some cases at least the inoculation of fresh bile could convey Rinderpest.

Before attempting to explain this I would say that, admitting for the moment that it can do so, the fact remains nevertheless that the method has been on the whole of incalculable service.

In Basutoland the inoculation with fresh bile was carried out by my veterinary assistant, Mr. Robertson, M.R.C.V.S., and Mr. Armstrong, M.R.C.V.S., assistant to the Colonial Veterinary Surgeon.

The inoculations made amounted to about 100,000 head of cattle, of which some 70,000 head were, for some considerable time at least, protected. No more successful results in the field have been achieved with this method than these, and it is of interest to note that the bile used was in every case obtained from *animals that had acquired the disease spontaneously* and had died only very shortly before the bile was collected. Thus the animals died at the termination of what may be called the natural period of the disease. In animals inoculated subcutaneously with blood the period of the death is irregular and very frequently if the animal is allowed to die the bile is unfit for use, but if killed at an earlier stage it is yet green in colour, and in this respect conforms to what is required. *Such bile does not always immunize.* In my own experience I have found that animals inoculated with such a bile, green and sweet smelling, were not immunized so that an inoculation of 0·2 c.c. of virulent blood ten days later set up virulent Rinderpest from which they died.

Dr. Koch shewed that the addition of virulent blood to ordinary bile (from healthy animals) if well mixed and injected into healthy animal did not produce Rinderpest but did not give any immunity. He further shewed that the addition of Rinderpest blood to good Rinderpest bile did not produce Rinderpest, and he was of the opinion that the addition of blood actually increased the immunising power.

I have shewn by the results obtained from glycerinated bile, that bile possesses an immunising property possibly due to the previous action of but not dependent upon the presence of living Rinderpest microbes.

Dr. Koch believed that good bile possessed an inhibiting substance which acting on living germs in the bile prevented them from setting up the disease. In a bile taken late on in the period of the disease (in blood inoculated animals) the bile is considerably altered even to the naked eye, and while glycerination has admitted of proof, that the immunising substance is present in great amount, I have found that inoculation of animals with such bile is in some cases attended with the subsequent onset of Rinderpest. With such data before me I consider that it is possible to understand how bile may, under certain circumstances produce Rinderpest, for if an early bile fails in regard to possessing immunising property, and a late bile, while possessing immunising power has lost its inhibiting substance, it is clear that Rinderpest may be produced.

I have already shewn that the admixture of virulent blood with glycerinated bile produces Rinderpest, even when so small a quantity as 0·1 c.c. is added to 20 c.c. of the glycerinated bile. For the purpose of obtaining bile I am opposed to the infection being made by the subcutaneous injection of blood, and I have a decided preference for the method of infecting either by smearing the nose with virulent mucus or blood, or by drenching the animals with blood diluted in water. According to the recent researches of Fraser, the bile of all animals has peculiar antitoxic virtues, and he suggests that the ingress of poison by the alimentary tract is attempted to be met with and antagonised by the bile secretion, so that the giving of Rinderpest to animals by *the natural channel* may, and I think probably does stimulate bile production, at least up to a certain period in the course of the disease, although my observations would seem to shew that towards the end of the period of disease bile secretion is almost entirely arrested.

Further, where infection is made through the gastro intestinal tract, the poison must in the first instance act through the portal vein, while in subcutaneous infection the virus must be carried to the liver, for the most part along the branches of the hepatic artery, a most important difference, seeing that in the one case the action proceeds, with regard to the hepatic lobules, centripetally, while in the other case it proceeds centrifugally. Were it necessary to again have recourse to the preparation of bile at large stations, I would strongly advocate the infection of the cattle being made by smearing the nose or by drenching with mucus or blood. I must express my conviction that *if bile were taken at a late period as Dr. Koch has recommended*, and from *animals spontaneously infected*, and if further, the bile were selected having regard to the qualifications as stated by Dr. Koch, I do not think the onset of Rinderpest would be so commonly found to follow upon inoculation.

At the present moment many farmers having inoculated first with glycerinated bile are following it up with periodic inoculations of fresh bile. In other cases they are using inoculations of glycerinated bile only, once a month, and they maintain that they can ensure that their cattle will remain free from the disease.

The main objection, however, to the bile system of inoculation is that although the cattle may remain for a few months immune, they will eventually become again susceptible unless periodic inoculations of virulent blood are made for some weeks after the bile inoculation, so as to produce some reaction, and if serum is procurable, one can force the reaction with considerable safety.

Serum Inoculation —Dr. Koch showed that the serum of the blood of an animal which had suffered from Rinderpest and recovered possessed immunising or protective power. He inoculated an animal with 100 cubic centimetres of such serum and on the following day inoculated it with 0·5 c.c. of virulent blood. After it had remained well for six days it was again inoculated with a larger dose of virulent blood, viz., 1 cubic centimetre. As the animal withstood this second inoculation, Dr. Koch concluded that it was clearly demonstrated that such serum possessed a certain protective power, but he felt and stated that further experiments would be requisite before he would be able to say for how long a period such immunity would last.

He also found that if to such serum 1 per cent. of virulent blood was added, and if during twelve hours subsequent the mixture was kept in a cool place and shaken up from time to time, that an injection of 20 cubic centimetres sufficed to induce a measure of immunity which could be afterwards increased by means of further blood injections.

It has been abundantly proved that the serum from animals which have recovered from Rinderpest is not possessed of the same immunising power in every case, and with a view of arriving at more constant results, Drs. Danyz and Bordet found it of advantage to reinoculate "salted" animals with large doses of virulent Rinderpest blood. They found that by this means the immunising power of the serum was considerably raised, and the serum of each animal was brought somewhat nearer a common standard. Their method, of which more may be said later, consisted in the use not of the separated serum, but of defibrinated blood, which may be looked upon as serum in a crude state, consisting, as it does, of serum and some of the blood corpuscles

The method by which the serum is made more strongly immunising—namely, by subsequent injection of virulent blood subcutaneously — is spoken of as "fortifying," and such serum may be referred to as "fortified" serum in contradistinction to the serum drawn simply from recovered animals, and which may be spoken of as "salted" serum.

Drs. Turner and Kolle have adopted the method of "fortification," which they have carried out to a high degree in using doses of several thousand cubic centimetres for the subcutaneous injection of animals. In carrying out such a method it is of course to be understood that one begins with small doses which are progressively increased. Furthermore, they suggest that the use of serum is better than defibrinated blood because it can be more easily conserved, while the mixing up of the serum from a number of animals gives more uniformity of strength, and admits of an attempt being made at standardization.

In such a disease as Rinderpest it is obviously impossible to arrive at any exact standard, because in order to test the serum for standardizing purposes it is necessary to have a standardized material to test against. In this case Dr. Kolle has suggested the use of one cubic centimetre of defibrinated virulent blood which is injected into one side of an animal, while 20 cubic centimetres of serum injected on the other side should prevent the action of the contagium of the virulent blood from setting up Rinderpest in a virulent form.

As, however, the virulence of blood is not always the same, it follows that such a method cannot be taken as giving absolutely exact data, but if the blood is taken from animals that have been injected with virulent blood, and if such a strain is being constantly maintained, then such blood, if not furnishing a standard infecting medium, is yet the nearest to such that is at present attainable.

After the Rinderpest had advanced through the Transkei and arrived at Alice, and inoculation was in progress in the Eastern Province, my further work was carried out at the Institute at Grahamstown.

Facility being thus furnished me for exact experimentation, I have endeavoured to determine in what manner the various immunising materials act in regard to the contagium of Rinderpest and in relation to each other.

In the first place I was anxious to determine in what manner the immunity was induced which had been granted by the use of glycerinated blood.

The question which obviously demanded my attention was whether it had been due to a modified or attenuated form of the contagium or to a chemical substance contained in the blood and produced either by the microbes themselves or by the body under the stimulus of microbic action.

In order to determine this more clearly I received virulent blood into a sterile solution of the citrate of potash and boracic acid, the latter being present in the solution to saturation.

Having obtained such blood it was kept for several weeks at ordinary room temperature and tested on animals to determine in a satisfactory manner that all virulence had disappeared.

Two animals were then inoculated subcutaneously with 30 cubic centimetres each on the 29th September. Ten days later each received 0·1 c.c. of virulent blood, the virulence being proved by the inoculation of two other animals which were inoculated with the same blood. After another ten days had elapsed, during which no rise of temperature had occurred while the two control animals had developed Rinderpest, they were again inoculated with a much larger dose, namely, 1 c.c. of virulent blood, but Rinderpest was not produced.

At the same time (September 29th) Nos. 176 and 177 were each inoculated with glycerinated bile, and after ten days with 0·1 c.c. virulent blood.

After a further period of ten days, no rise of temperature having been produced, they were again inoculated with 1·0 c.c. virulent blood, but without producing Rinderpest.

In the same way No. 167 was inoculated with 20 c.c. fortified serum, and subsequently with 0 1 c.c. and 1·0 c.c. of virulent blood, periods of ten days being allowed to intervene between the inoculations. On the 29th September also, Nos. 174 and 175 were each inoculated with 10 c.c. glycerinated bile and 10 c.c. blood (kept in citrate of potash and boracic acid), the blood and bile being thoroughly mixed before inoculation. Ten days later each animal received subcutaneously 0·1 c.c. virulent blood, and, no rise following, each received ten days later 1 0 c.c. of virulent blood, without Rinderpest being produced.

Also on the 29th September Nos. 170 and 171 were each inoculated with 20 c.c. fortified serum and 20 c.c. blood (kept in citrate of potash and boracic acid).

On the following day No. 170 received 0·1 c.c· virulent blood. Nine days later in the case of this animal, and ten days later in the case of No. 171, each was inoculated with 0·1 c.c. virulent blood, and no rise of temperature following meanwhile, each was again inoculated with 1·0 c c. viruleut blood without giving rise to Rinderpest.

Also on the 29th September, Nos. 168 and 169 were each inoculated with 20 c.c. of fortified serum and 20 c.c. of glycerinated bile. On the following day No. 168 received 0·1 c.c. virulent blood, and nine days later in the case of this animal and ten days later in the case of No. 169 each received 0·1 c.c. of virulent blood. Ten days later each received 1·0 c.c. virulent blood, but Rinderpest was not produced as a result of this inoculation.

It will thus be seen that on the same date the following inoculations had been made :—

Two animals with non-virulent blood 30 c.c.
,, ,, fortified serum 20 c.c.
,, ,, glycerinated bile 20 c.c.
,, ,, non-virulent blood and serum 20 c.c. ea.
,, ,, glycerinated bile and serum 20 c.c. each.
,, ,, ,, ,, blood 20 c.c. each.

Ten days later each was inoculated with 0·1 c.c. virulent blood, and after another interval of ten days had been completed they were again inoculated with 1·0 c c. of virulent blood.

In each case the different animals were inoculated with the same sample of virulent blood whose virulence was proved by inoculating clean cattle with it at the same time.

The object in view was to determine if any of the immunising materials in any way antagonized the other, and as the immunity which follows serum inoculation comes on early I inoculated one animal of each series in which serum was used on the following day with virulent blood, considering that if the other material, whether glycerinated bile or non-virulent blood, exerted or could exert any antagonistic action, it would have the effect of interfering with the serum and hence allow of Rinderpest being produced by the inoculation of virulent blood on the following day. As subsequent events proved, however, the mixtures of the various immunising materials succeeded in giving rise to immunity as completely as either material did when used by itself.

I therefore concluded that neither Rinderpest blood (kept in such a manner as to allow of its virulence becoming destroyed) nor glycerinated bile, nor fortified serum acted in any way antagonistic to the other. Before proceeding further I considered it necessary to determine what substances in non-virulent Rinderpest blood had immunising property. That is to say, whether it was in the serum, or was contained in the particulate components of the blood. To determine this, two animals which were sick with Rinderpest, and approaching death, were bled into sterile glass jars, and the blood then set aside to admit of the serum separating. Such blood, I find, does not coagulate either quickly or firmly in all cases, and the serum which separates tends to be heavily tinged with hæmoglobin.

In this case, however, a fair amount of serum separated, which was carefully decanted, and after filtration through paper, was again filtered through a Pasteur filter. Such filtered serum may be referred to as "sick serum" in contradistinction to "salted" and "fortified."

Obviously, if the use of glycerinated blood in a manner similar to glycerinated bile can give rise to a weak passive immunity, the immunising agent might be expected to be present in the serum of such blood.

Animals No. 211 and 212 were inoculated with 30 c.c. each of "sick" serum on the 26th October. On the following day No. 211 received 0·1 c.c. of virulent Rinderpest blood. During the nine days which followed no rise of temperature occurred, and it was accordingly inoculated with 1 c.c. of virulent blood. On the following day the temperature rose to 103·3 in the morning, and fell the same evening to normal. Three days later a sudden rise occurred to 104·4, but immediately fell to normal. During the next three days the temperature was normal. Two days later the animal died suddenly in the early morning during a severe cold rainstorm. No evidence of Rinderpest could be found. No. 212, which had been inoculated on the 26th October with 30 c.c. "sick" serum, was re-inoculated ten days later with 0·2 c.c. of virulent blood. Four days later the temperature rose to 105, but at once fell and remained low all the following day. No Rinderpest occurred.

No. 213 was inoculated on the 26th October with a mixture of 20 c.c. "sick" serum and one cubic centimetre of virulent blood.

The mixture was kept at ordinary room temperature for 24 hours. Twenty one days later, no rise of temperature above 103·3 having meanwhile occurred, it was inoculated on the 16th September with 1 c.c. of virulent blood. The temperature rose on the following day to 104 in the morning, and 107·4 in the evening, after which it fell again to the normal and remained so despite the fact that it was run among the sick cattle of the infected camp.

No. 242 was inoculated on December 18th with 30 c.c. "sick" serum, and one hour later was inoculated with 0·5 c.c. virulent blood. On the sixth day the temperature rose in the evening. On the seventh day to 104·2 in the evening. After the temperature had remained high during four days 100 c.c. of fortified serum was given.

The animal made easy recovery, and has since been inoculated with 2 c.c. virulent blood without any appreciable result.

No 246 was inoculated on December 18th with a mixture of 25 c.c. of "sick" serum and 5 c.c. "fortified" serum. One hour later it received 0·5 c.c. virulent blood: On the fourth evening the temperature rose to 104, on the next to 103·4, next 105·3, then fell to normal. Three days later a sudden rise to 106 seeming to suggest that the modified attack was passing into a virulent one, led me to give a dose of fortified serum, but I do not now think it was necessary. The animal made a good recovery.

It is therefore abundantly proved that in the serum of the blood of animals affected with Rinderpest, and at a period which may be stated as at the height of the fever, there exists a substance which possesses immunising properties, but it is also evident that the immunity so produced is weak since it can readily be upset by a moderately strong dose of virulent blood.

Taking into consideration that a dose of 30 c.c. can give an immunity after 24 hours sufficient to admit of resistance being given to 0·1 c.c. of virulent blood, one might naturally expect to find that it could exert some curative action.

Experiment, however, shews that sick serum, when used to inoculate an animal already sick, has very little curative effect, although it is clear from inoculations made, whereby 0·5 c.c. virulent blood was first inoculated and followed an hour later by 30 c.c. of "sick" serum on the opposite side and a modified attack resulted, that a distinctly inhibitary action is shown on the progress of the disease. Also, since a mixture of 25 c.c. of sick serum and 5 c.c. of fortified serum inoculated and followed by 0·5 c c. virulent blood also produced a similarly modified attack, it would seem that "sick" serum does not antagonise the action of "fortified" serum. From our experience of the antitoxin of diphtheria we know that the toxin of the diphtheria bacilli antagonizes or is antagonized by the serum of the blood of horses "salted" or immunised against the action of the toxin, hence if a toxin is present in the blood of Rinderpest animals, it must be concluded that the serum of immune animals does not contain any anti-toxic substance. On the other hand, if a small amount of Rinderpest blood (1· c.c.) is mixed with 20 c.c. of fortified serum and kept at ordinary room temperature for 24 hours, the subsequent inoculation of the mixture does not give rise to Rinderpest at all, or if the serum is simply "salted" serum and weak enough not to destroy the contagium, the disease produced will be modified in character. I therefore conclude that the serum of immunised animals contains a microbicidal substance.

Assuming for the moment that my conclusion is correct, it remains to be considered in what manner the other non-infective substances, such as glycerinated bile, glycerinated virulent blood, and serum of Rinderpest blood differ in their action from the serum of immune animals.

In many experiments where I have used such substances for the purpose of inducing immunity, and where at a later date virulent blood has been used in such a dose as nearly sufficed to overturn the immunity and produce Rinderpest, I have been struck with the fact that on the evening of the same day on which the virulent blood had been inoculated, or at the latest next day, there has been produced a sharp and decided rise of temperature which almost as quickly falls. Now, as such a rise has invariably occurred at too early a period after inoculation to be considered as a true febrile rise consequent on a period of incubation, it has seemed to me as if the action was due to the stimulus of a material contained in the blood acting upon or in succession to the material already present in the system as the result of the former inoculation with non-infective material.

In the case of animals already suffering from Tuberculosis or Glanders, though no symptoms may be present, we know that the inoculation of a small dose of non-infective toxin produces in most cases a sharp rise of temperature which is diagnostic and is accepted as proof that the animal is already affected by the disease. In such cases we have, firstly, in the system the contagium, together with the toxin which it has elaborated, and, secondly, there is added a dose of toxin, which, upsetting the equilibrium, gives rise to the effect of a febrile reaction. In the case of Rinderpest, however, we have the equation reversed; firstly, we have a dose of toxin in the system, and there is subsequently added a small amount of living contagium, together with some toxin formed by it, and the result is similarly seen in the sudden sharp rise of temperature which follows.

Apart from this there is the fact that the strength of immunizing serum is increased by giving large injections of virulent blood, and as we know that in an immunized animal the specific microbes remain in the part into which they are injected, and are very quickly destroyed, hence any action that results from large doses of blood can scarcely be considered as arising from vital microbic action.

In the case of bile, however, I cannot but be impressed by the fact that the immunity produced by its action persists in greater or lesser degree over a much longer period than is the case where glycerinated blood or sick serum is used. It is possible that the bile may contain some special substance which is antagonistic to the contagium of Rinderpest. Professor Fraser has already discovered that the bile of serpents contains an antivenomous substance, and that a similar substance or substances, though in lesser degree, are found in other biles. Admitting this as probable, it would, in my opinion, serve to account for the difference of the immunizing value of bile as compared with other substances.

I am therefore of opinion that the non-infective Rinderpest materials, to which I have already referred, produce weak or passive immunity by virtue of a toxic action on the blood or tissues, which in turn stimulates the system to the production of specific microbicidal substances. Under, or in conformity with such a line of argument, we should expect passive immunity to be produced in proportion to the dose or number of times a dose of non-infective material had been injected. I have amply proved this to be so, for if a dose of glycerinated bile be given to an animal and repeated after an interval of ten days, the immunity is so great that 0·2 c.c. of the most virulent blood can be given, and followed after periods of eight days by doses of virulent blood, each being ten times the quantity of the preceding, with nearly absolute safety.

Methods of using Immunising Serum.—The use of a dose of serum is largely dependent upon the strength of the material. For a high degree of fortification it is usual to inoculate animals which have been immunised or have recovered from the disease, with progressively increasing doses of virulent blood, and it is found that by so doing the immunising strength of the serum becomes gradually increased. Such a serum is always more reliable than that of simply "salted" cattle. In the latter case the serum is most variable, for in a case in which an animal has suffered from a mild attack, 60 c.c. may be required to antagonize by simultaneous inoculation 1 c.c. of virulent blood. On the other hand, if an animal has just recovered from a mild attack, and if it be immediately inoculated by blood injection (where the former was spontaneous) or by nasal inoculation with virulent mucus (where the former attack was due to blood injection), a second attack will supervene, more or less severe in proportion to the mildness of the former one.

In such cases I have found that if after the temperature which had been raised in the second attack is permitted to regain the normal, that the serum drawn from this animal's blood six days later has an exceedingly high immunising value, so that in the cases I have investigated a dose of less than 30 c.c. would antagonise the action of 1 c.c. of virulent blood which was inoculated simultaneously. I also smeared a healthy ox and a young heifer over the nose and mouth with a plentiful supply of virulent nasal mucus from a typical case. On the following day it was inoculated with 30 c.c. of this serum, but the temperature during the following three weeks was very little above the normal. It was then inoculated with 1 c.c. of virulent blood. On the evening of the fourth day the temperature rose to 104; was 105 on the following day; 106·8 on the day after, and then immediately fell to normal. The heifer which was smeared with the nasal mucus died of Rinderpest.

This serum also possessed high curative powers as tested by its action on animals suffering from the disease with high temperature. In the preparation of a fortified serum where large doses of virulent blood (one litre or more) are being used for inoculation, it is necessary in order to obtain the best curative action to permit the animal to rest for eight or nine days. I then prefer to give an intravenous injection of 100 cubic centimetres of the most virulent blood I can obtain, and to again wait nine days, because I have found that commonly a late reaction of temperature occurs, following upon this comparatively small dose, about the seventh or eighth day. By this means it is possible to obtain a serum of which a little less than 15 c.c. serves to antagonise the action of 1 c.c. of virulent blood.

In using serum simultaneously with virulent blood for the immunisation of healthy cattle, either by:—

1. Inoculating 100 c.c. of defibrinated blood on one side, and 10 c.c. of virulent blood on the other.

2. Or by the more exact method suggested by Drs. Turner & Kolle, by which one cubic centimetre of virulent blood, being injected into one side of an animal, was to be followed immediately by the injection of 20 c.c. of a standardized serum on the other side.
3. Or by injecting a small dose of virulent blood, and after waiting some hours or days, injecting serum.

All such methods are open to the disadvantage that if spontaneous infection obtains access to the herd previous to the action of the virulent blood being completed, disaster may follow. The French experts, Drs. Danyz and Bordet, endeavoured to evolve a feasible method of inoculation by the farmer by injecting a serum which was not highly fortified, and then exposing the animals to spontaneous infection among sick cattle. This method has undoubtedly worked well in their hands, but as the serum of some animals is, as I have shewn, very highly immunising, while others are comparatively weak, the effect has been in many cases that they were so quickly immunised that they resisted infection, and after the immunity passes, which it does in from eight to twelve days from a small dose, they may thereafter take the disease severely.

The objection to simultaneous inoculation of blood and standardized serum is, in my experience, that though some animals will take the disease mildly or more severely, others in the same batch may not shew a rise of temperature, whether due to their less susceptibility or quicker absorption I cannot say.

Where, however, the temperatures of the animals are regularly taken, this method works well, because more serum can be given to those in which the disease is evidently progressing too violently, and where the febrile reaction lags we can at a later date give a second blood inoculation.

To all the blood inoculations, however, there is the almost inseparable objection that the blood of a Rinderpest animal may convey another disease in addition to Rinderpest.

Drs. Turner and Kolle have already found this to be so, and I have had an experience which is further referred to in connection with Redwater or Texas Fever. I have found by experience, and corroborated by scientific experiment, that the blood of an animal which has been acclimatized to a Redwater area, although it may not itself give any evidence by symptoms, nor its blood on microscopical examination shew the presence of the parasite, may when inoculated into an animal susceptible to Redwater give rise to this disease in a more or less fatal form. The parasite will be found present in the blood of the animals becoming so affected, but, strange to say, the parasite does not conform to the typical form as seen in unmodified Redwater or Texas Fever. In order to overcome this difficulty I have endeavoured to produce Rinderpest by drenching with virulent material, and to control the febrile reaction by serum. In carrying this method out I have added one quart bottle of virulent blood to a pailful of water (dilution is thus about one part of blood to thirty of water). Each animal is then drenched with a third of a quart (it is not necessary to be absolutely exact). After three days each animal should receive a dose of serum 50—100 c.c.'s, then one waits until the temperature begins to rise, and immediately gives each one which has any rise a dose of serum (50 c.c.) daily till the temperature falls. Should any fail to react, they can be drenched again without much risk. Out of six so treated, all did extremely well, making good recovery, excepting one cow which had a dead calf in the uterus, and which died from other complications, it being, as a matter of fact, recovered from Rinderpest.

In using serum for immunisation, the principle to be adopted is to endeavour to produce a febrile rise of temperature in the presence of a small amount of serum in the system, and when the fever has been produced, to arrest it by the curative action of further doses of serum.

Care, however, must be had that the febrile change must not be too quickly arrested, else the immunity is not high. I prefer to see the temperature remaining high over two or three days while serum is being administered, and where any doubt as to immunity remains, to drench the animals with virulent blood at a later date, about a week after the fall of the temperature to normal.

In most cases where a second infection is made, the resulting disease is readily amenable to serum.

APPENDIX 4.—THE QUESTION OF THE RELATIONSHIP OF RINDERPEST TO VARIOLA (SMALL-POX).

Among the earliest records of descriptions of and enquiries into the disease known as Rinderpest, there appear statements in which it has been affirmed that Rinderpest was variola of the ox. This belief was first opposed by Professor Novara, of Patavia, in 1711. Nevertheless, this theory has been from time to time revived, and during the investigations of the Commissioners appointed in 1865 to investigate into the nature of Rinderpest, experiments were conducted with the object of deciding this point, and whether, as stated, the practice of vaccination with vaccine lymph afforded any protection against the disease. Dr. Sanderson experimented by inoculating a number of animals with Rinderpest virus, after they had previously been successfully vaccinated with vaccine lymph. He concluded that the progress of the Rinderpest in these cases was such as to lead him to consider that it had not been in any way modified by the previous vaccination.

Dr. Murchison, also one of the Commissioners, stated that: "The resemblance between Rinderpest and variola is sufficiently striking as to demand immediate enquiry. . . . . If the reply to the enquiry be in the affirmative, a certain method of arresting the spread of the cattle plague will be placed in our hands."

This gentleman later expressed his belief that vaccination did give protection, but not of a lasting character. He also records notes of a case in which a gentleman who was making an autopsy on an animal dead of Rinderpest received a scratch which later developed into a pustule that corresponded with what is found in cow-pox.

During the progress of my work in connection with Rinderpest my assistants and myself have had ample opportunities, during the making of autopsies and other operations on Rinderpest cattle, of becoming inoculated with the virus, but excepting that on one occasion a scratch which I had received on my hand and was overlooked, gave rise to local swelling, followed by local swelling of the lymphatics of the arm and enormous tumefaction of the axillary glands, with accompanying fever, no local lesion has been produced so far as I am aware on the human subject as a result of inoculation with Rinderpest virus. It is true one gentleman died who contracted a cut during the skinning of an animal, but in that case, as in my own, there is no evidence to shew that the poisoning was other than a septic poisoning which might, under similar circumstances, arise from an inoculation wound when working with a carcase from an animal dead of any disease.

Seeing that this Institute is concerned with the preparation of vaccine lymph, and that we do not as a rule destroy the cattle used in the production (tuberculosis in cattle being practically unknown in this country), I felt that the enquiry would be of great practical as well as scientific importance to us. In the first place, should vaccination not modify Rinderpest, then the cattle might be thereafter used for the production of Rinderpest blood which was necessary for the fortification of immunised animals, whose serum would be afterwards used for curative purposes.

Briefly, then, I may state that during a succession of months I have used such cattle that had already yielded lymph which gave successful vaccinations on the human subject, but I have not been able to find that the slightest protection against nor the slightest modification of Rinderpest has been produced by vaccination with lymph.

During the period when bile inoculation was being pushed on in the country around, it was very difficult, indeed, well nigh impossible, to procure calves for vaccine production. I had, however, nearly 500 animals in my experimental camp which had suffered from Rinderpest in a severe form and recovered. Among these were a considerable number of young animals which might suit for vaccine production. Some of these had received injections of Rinderpest blood in doses which in some cases were as large as 1,000 c.c.

I attempted the vaccination of these cattle with good lymph, and succeeded admirably. This strain of lymph was carried through more than forty animals. It was never modified in any way, and produced, in other hands and my own, typical results both in primary and re-vaccinations, and from one such vaccination I made a retro-vaccination successfully.

I am therefore of opinion that no relationship whatever exists between vaccinia and Rinderpest, and that neither, affecting an animal previously, induces any modification in the other if subsequently induced.

ANNEXURE.

Historical Retrospect of Rinderpest.

In the following account, I am largely indebted to the admirable works by Fleming (Animal Plagues) and to Gamgee's work on the Cattle Plague. With regard to occurrences of this malady during the period which has elapsed since 1865, the year of the great cattle plague, authoritative data to which I have access are very scanty and I have been forced to draw what information I can bring forward from the somewhat brief accounts that have occurred in contemporary literature.

The first authentic account of what was in all probability the cattle plague had reference to the year A.D. 69. Columella, who lived at this time, and who, according to accounts, had considerable influence on the progress of Veterinary medicine, has written of many of the diseases then current among lower animals

In the ninth chapter of the sixth book, he says:—"When an ox has the fever—the fever referred to being probably Rinderpest—he must not be allowed to eat for the space of a day, after which, and before he has eaten anything, a little blood must be abstracted from under the tail, then he must be made to swallow thirty cabbage leaves baked in oil. This food is to be given for five days, and while fasting, and he is to have, besides, the tops of the lentisk, the olive tree, or any other species of tender leaved plants, or branches of the vine, and cold water is to be allowed him to drink three times a day." He further adds that "this treatment ought to be followed out in the cowhouse, and the animal should not be allowed in the open air until cured."

The symptoms of the fever were as follows:—"The fever is present when tears are running down the face, when the head is carried low and heavily and the eyes are closed, when the saliva flows from the mouth, when the respiration is shorter than in health and seemingly embarassed or sometimes accompanied by groaning." He further insisted on the most sensible procedure of separating the sick from the healthy stock.

A.D. 376.—In the East of Europe, a very severe epidemic arose and began to spread westward. It was of an exceedingly virulent character and occasioned enormous loss as the cattle seemed to die almost as soon as they became sick.

All attempts at cure proved unavailing and from its course and virulence there is little doubt but that it was the true cattle plague.

During the height of the epidemic, no animal was deemed safe unless branded in the forehead with a red hot iron in the shape of a cross.

A.D. 381.—Vegetius Renatius, a skilled agriculturist of the time, describes what he calls the Malis or Mallens and which Fleming thinks may have been the cattle plague.

He says:—"All these diseases are very contagious, and if one animal be seized by them they pass immediately to all, and so they bring destruction upon whole herds or upon all those that are fully domesticated and trained to labour. Therefore it is that animals which have been attacked must, with all diligence and care, be separated from the herd, put apart by themselves and sent to those places where no animal is pastured, lest by their contagion they endanger all the rest, and the negligence of the owner be imputed (as is usually done by fools) to the Divine displeasure.

A.D. 591.—During this year Italy was invaded by a terrible plague of locusts which were supposed to have come from Africa. The devastation caused by them was terrible, every green leaf or herb being eaten up. Being eventually blown into the sea they were again washed up on shore and the putrefaction which followed was stated to be the cause of an enormous fatality in men and beasts by which nearly a million perished. In France and Belgium, following upon famine and plague there arose "a grievous plague amongst cattle and oxen, which increasing, left scarcely any to breed from. Not only did this plague affect the domestic animals—it also attacked wild creatures. The remains of a multitude of stags and other beasts were found dead in the forests."

In the year A.D. 694 a great plague attacked the cattle in England, causing tremendous distress. The following year it invaded Ireland and with the famine that existed for three years, destitution was so great "that men and women did eat one another for want" (Annals of Clonmacnoise).

During the year A.D. 801, a great pestilence occurred among the cattle in France, the origin of which was attributed by malicious people to Grimoald, Duke of Benevento who, it was stated, hated the Christian king and sent emissaries with enchanted powders to sprinkle over the land with the intention of killing the animals The heavy mortality which occurred suggests that the epidemic was Rinderpest, but as Fleming remarks "it would seem that from the time of Thucydides to the present day, when a strange disease suddenly appeared, the masses have always entertained suspicions as to its mortal effects being due to poisonous substances introduced into the water, food or air by malicious people."

A.D. 809-10.—Again a great epidemic among cattle arose in Eastern Europe and passed westwards which was probably Rinderpest. Fleming also believes that Rinderpest was at this time in Britain. The archives of the Imperial Agricultural Society of Russia, state that at this period the pest was imported from the Asian shores of the Black Sea into Europe. It spread to Hungary, Germany, Austria, Flanders, and was probably then carried to England. The mortality was enormous.

The spread of the malady at this time has been ascribed to the wars which were then occurring.

A.D. 820.—Rinderpest appeared in Hungary and after raging with intense virulence passed westward.

A.D. 850.—During this year the Rinderpest appeared in France and in many provinces almost the entire horned stock was swept away.

A.D. 940.—In France, Italy and Germany there occurred an epidemic among cattle which was probably Rinderpest.

A.D. 1223-25 —During this century up to the year 1241, Mongol invasions to Silesia through Russia occurred, and as it has been justly conjectured many epidemics were introduced into the western hemisphere. The epizootics of 1222, 1233 and 1238 are believed to have been Rinderpest, which were in this way conveyed westward. The epidemic of 1223 caused an enormous mortality among cattle of which the greater part died.

In 1240 Rinderpest appeared in Hungary and thereafter invaded nearly the whole of Europe.

From the beginning of the 18th century the history of epizootic diseases becomes more exact. Owing to the great national interests involved the diseases of the lower animals was now receiving more careful attention, and from this date researches of great importance were devoted to the study of this branch of comparative pathology. As a result of this we find carefully detailed descriptions of epizootic diseases and reports shewing the amount of careful observation which was made into the nature, origin and best means of prevention of diseases. Schools for the study of Veterinary medicine now became established of which the first was established at Lyons through the instrumentality of M. Bourgelat in 1762. In the following year the French Government established the Veterinary College now at Alfort, near Paris. A third was soon instituted at Toulouse, and soon afterwards Vienna established a National College by order of Maria Theresa, which being remodelled and reorganized by Joseph II., was one of the largest in Germany.

In 1768 a school was founded at Berlin through the influence of Cothenins, but the idea would seem to have originated with King Frederick the Great. Thereafter schools sprang up in Germany, Holland, Switzerland, Denmark, Sweden and Russia, and through the patient exertions of a private nature a school was established in the Scottish Capital.

A.D. 1708.—In this year Kanold asserts that Rinderpest had commenced its destruction in Russia. The beginning of the plague in Europe was at that part of Tartary near the border of Asia, and it is a question whether it did not arise from this latter country. It was very contagious, affecting large numbers of cattle of which most died almost at once, and it extended over many kingdoms steadily and uniformly notwithstanding all care. In 1710 it was spreading in Russia, in the Governments of Riazan, Worston and Moscow, and in the autumn had reached to Ukraine, and was spreading in Volhynia, Podolia and Transylvania. In the year following it spread from Russia through Poland into

Austria, Bavaria and Suabia, from Dalmatia into Italy, and from Russia into Moldavia and Wallachia. From Moscow, Riazan, Worsten, Ukraine, Podolia and Volhynia it passed to Polesia, Lithuania, Sendomer, Lublin Cracow, Siradin, Kalisch, Posen, Masovie and entered Russia arriving in October at Königsberg; from the other side it passed to Silesia and became diffused near Bojanowa, Medzibor, Ohlaw, Brieg and Breslau It spread only by contagion, and Kanold says of it that "notwithstanding the lateness of the season they who had two or three herds of stock on their lands could scarce keep a single animal, and large numbers of cattle were found lying dead upon the roads." From Hungary it passed to Styria, Corinthia, Austria and Augsburg in Bavaria. Everyone agreed at this time the disease was being spread by infected cattle which were being driven from Hungary into Italy. Gerbezius, however, attributed its origin to the state of the pastures where, from the excessive rains, the numerous locusts and cicado were undergoing putrid decomposition.

It was stated that the State of Carniola avoided the pest through prohibiting the admission of pigs from Crotia. According to Schroeckins "about the end of the summer and throughout the autumn that plague which had been so destructive to the bovine race in Germany and Italy after it had proceeded by degrees from Hungary towards the Danube, attacked our territory and produced great destruction to beasts, sometimes destroying whole herds amongst us, and in many neighbouring places. And this was not caused by any foulness in the atmosphere, but by the contagion of oxen brought from the infected countries; and this was latent, because it first attacked those pastures adjoining the foreigners, and altogether spared those cattle to which no infected animals had approached, and which had been immediately separated from any in the same herd which were infected. The saliva of the diseased beasts dropped in the pastures affected them, and thus communicated the malady to those cattle which afterwards grazed thereon.

It appeared certain that this acrid matter passed by way of the mouth, œsophagus, the stomachs, and the bowels, and in infecting these, caused an irritation which was soon communicated to the nerves from whence arose the spasmodic movements. The constriction of the vessels which followed, induced congestions and inflammations and converted the whole body into a corrupt mass. In some the tongue was inflamed and covered with red vesicles, the excretions were sanguinolent, as in malignant dysentery. The principle symptoms of the disease, at the commencement, was a difficulty in breathing and a higher temperature than in health. The intestines near the liver were found after death covered with bile and the stomachs inflamed." In Fleming's account he says, "The disease spread from Russia and Hungary into Moldavia, Wallachia, Sclavonia, Istria and Dalmatia." and as already stated it had entered Italy from Hungary.

Fleming recounts its history after the infection had reached Padua, "On the 27th of August, 1711, a drove of infected cattle from Hungary, sent through the agency of Dalmatian merchants and which had been disembarked at Venice passed through the village of Sermiola, about two leagues distance from Padua, and one of these beasts straying from the others, was taken to a farm named Pampagnini, belonging to the brothers Barromes, where it was put into one of the cowhouses. In about eight days the whole of the cattle on this farm became ill, and soon all died, with the exception of one which had been treated by a seton in the neck. The disease soon spread into the neighbouring districts, and it was believed, and the public records sanctioned this belief, that this Hungarian ox had conveyed the germs of the pestilence."

Of all the descriptions at this date relating to the nature and advent of the Cattle Plague probably the best are those of Lancisi and Ramazzinni two physicians who gave special attention to this disease.

Ramazzini gives a description in the following manner.—" It is evident that this disease, which has created such dreadful havoc among the whole bovine race, from its cold shiverings, followed by excessive heat throughout the whole body, is a malignant and deadly fever, as its accompanying symptoms testify. In the first place, there is intense anguish, heavy breathing and continued snorting (or groaning) accompanied by fever, stupor and slothfulness or uneasiness; a continued running of ill-odoured matter from the mouth and nostrils is observed, a most fetid excrement sometimes mixed with blood is passed; there is loss of appetite, and chewing the cud (rumination) ceases. On the fifth and sirth days pustules break out all over the whole body and tubercles of a variolous character. On the fifth or seventh day, death ensues which very few escape, and those more by chance than from the effects of remedies. We may reasonably suppose that the miserable oxen suffer much internal pain, when they lie groaning, or while they stand motionless with heads cast down towards the ground; but from dumb animals who can make no signs, it is impossible to say for certain what is the ailment in their case, and therefore remedies are difficult to find. In the carcases of as many oxen as were dissected by the eminent Professors Molinetto and Viscardo, it was noticed in all, strange to say, that the omasum was hard and compact, and the leaves closely adherent; while all were of great size and emitted a horrid stench. In other parts such as the brain and lungs, hydatids were found, enclosed in huge bladders as if full of wind; these, when dissected, exhaled a noxious effluvium. The tongue was covered with ulcers towards the root and full of little vesicles on its sides. I know for certain, that that portion of the body which was observed in the abdomen (the stomach) to be hard and compact like stones, was primarily produced by a contagious miasma, which while pursuing its own course of destruction weakened and corrupted the gastric juice."

Ramazzini with others concluded that the malady was similar to and identical with small-pox in man. This conclusion was discussed and made the subject of controversy in a circular which was issued by the

Philosophical College at Padua on the 28th October, 1711. This opinion curiously enough has from time to time been revivified and despite the fact of the experiments made and conclusions submitted by the English Commissioners in 1865 has still found support.

During the progress of my work in the past year this matter has engaged my attention and in the experimental Investigations recorded elsewhere in this report will be found an account of experiments which I think fully establish the conclusion that Rinderpest is in no way related to the disease known as small-pox in man.

The report of the Paduan College discussed the matter in the following manner: We have seen the effects of the disease to be most frequently in the viscera already described; that is to say in the first ventricle or omasum (the word ventricle is used in error, reference is clearly made to stomach) where one sees dryness, hardness, and contraction towards the middle, with a collection of alimentary substances rendered hard and stony; the second stomach is found extremely full of food, with an abundance of fetid gas. The following organic changes are found in the viscera, with few exceptions: We see the lungs evidently inflamed as well as the neighbouring parts; but at other times only the bronchial glands, tonsils, and adjoining textures, as well as the muscles of the œsophagus and larynx, the morbid appearances often extending to the tongue in a great number of cattle as a deep and somewhat transverse fissure, sometimes involving the whole organ; these fissures, as the disease progresses, become foul callous ulcers. Besides these various morbid characteristics, other rare (or inconstant) appearances are observed, such as suppurating tumours shewing themselves in the glands of the throat and abcesses in the lungs and liver; at other times a number of small tumours arise upon the skin covering the body which neither suppurate nor change colour but slowly disappear or remain until the death of the animal.

From this last and rare circumstance it has been the opinion of some that this affection should be universally designated *Variola Bovina*; to which disease, however, many high authorities thought oxen were not liable. But yet, if this be the case, as might be expected, how is it that in so general a disease as this variola is, so small a number exhibit the morbid eruption on the skin? Nor does variola usually make such great slaughter of the sick, nor yet is it so general or so rapid in its course." This was written by Marco Novara, professor of practical medicine, at Patavia.

Meanwhile the Rinderpest still existed in Russia, Poland, Silesia and Turkey. In Silesia 100,000 head of cattle had died. In Italy it caused widespread devastation. In Naples and the States of the Church 70,000 died during the month of September, while at Cremona 40,000 died. In Germany it still was spreading, and spreading widely through Bavaria and Saxony, reached Franconia.

During the year 1713 it still ravaged the countries already invaded in the preceding year, and in Russia it had effected an occupation of the Governments of Novgorod, Plaskow, Petersburg, and Ingermanland. In Germany there were numerous centres, as Bresgau, Wurtemburg, Baden, the Rhine Valley, Pfalz and Alsace. In Switzerland and Holland it was carrying widespread ruin. In the latter country 200,000 died.

In Italy its march was causing consternation and fear, and the learned physician to Pope Clement XI., Giovanni Lancisi, was sent to investigate the nature of the disease with the object of determining curative measures or methods of suppression. His report, so exact in detail, reflecting in so high a degree the peculiar ability of one who was, so to say, enlightened in advance of his age, cannot be overlooked as being nearly abreast of what we know at the present day. It reads as follows:—

> "In the middle of the summer of 1713 there was a rumour at Rome that a large number of infected oxen from the districts on the Mediterranean were being driven from the market of Frusino to us; wherefore it was wisely decreed that no market should be held nor any cattle admitted into the place. But merchants introduced oxen into the city

secretly by byways, because their hopes of selling them publicly had been frustrated; and these being driven about in all directions and becoming mixed with our hitherto healthy stock, spread abroad the disease. For when any foreign merchants had doubtful or suspicious cattle they could not sell in their own country they brought them to Rome surreptitiously and sold them for less than the usual price. But the butchers of the city, who had not yet discovered the deceit practised upon them, were led to buy these animals at a reasonable rate, and in this way were immediately disseminated the germs of the hidden contagion. Every fact clearly shows that the cause of the plague is some exceedingly fine and pernicious particles which pass from one body to another, by contact or by the means of fomites. It therefore resembles a special virulent poison: a few particles affecting the whole organism, and acting, in all likelihood, just as we see a ferment act in bread or wine, a minute portion leavening the entire mass.

The first symptoms of disease in some oxen we had under observation were timidity, bellowing, snorting, and other indications of sudden fright or agitation, as if some poison had affected their mobile spirit, causing spasms of their nervous fibres and producing convulsions externally and internally. We also saw others, but a few only, which were naturally weak and destitute of stamina, die suddenly as if struck by a thunderbolt. In general, however, the first symptoms of the plague were debility, sudden dullness, drooping of the head, tears flowing from the heavy eyes, and mucus and saliva from the nostrils and mouth. This was accompanied in the meanwhile by fever, with shivering and staring of the coat, nausea, and an inclination to lie down. There were always inflammations, pustules, hydatids and ulcers on the tongue and mouth, accompanied by intense fever.

In not a few there were scattered over the skin watery tubercules, and the hair fell off. At first they suffered from excessive thirst, and drank greedily. Afterwards they wholly abstained from food and water, and were unable to swallow or chew the cud. The want of sustenance caused death to take place more quickly than would have been the case from the nature of the disease. In the stomach lumbrici were very often found, and the dejections were very fœtid, of various colours, and sometimes stained with bloody humours. Nearly all the cattle, at the later stages, were foul-smelling; their breathing was laborious and heavy; not infrequently there was a cough, and within seven days they generally died. Those which reached another day—but they were very few—usually escaped, especially if the hair became rough and fell off. And if they could not easily arise from a recumbent position it was usual to prevent their attempting to do so.

It was proved by examination that they continually suffered from affections of the bowels. This appeared most evident on three examinations which we held on the dead bodies of oxen that had died of the disease, for besides ulcers in the mouth, fauces, œsophagus, stomach and lungs, there were spots of the colour varying from red to livid or gangrenous, nearly the same in each animal, but the lesions in the intestines were not alike in all. For in the first ox which had perished the third day we found in the omasum a hard mass of hay and that ball which Pliny calls *Tophus* of young heifers, produced by licking of the hairs with their tongue and rapidly swallowing them, when, by the peristaltic motion, they were rolled up, and the saliva was obliged to pass through as in a filter. The other intestines were in a fair, healthy state. In the second, which died on the sixth day, the liver, intestines and lungs were completely mortified. In the third, the heart and brain were nearly corrupt, and it is extremely wonderful that this deadly poison, which had destroyed the bovine race with such havoc in these years, was wholly innocuous to other animals. It is also remarkable that the seeds of the contagion were not only caused by sick oxen, but were more frequently conveyed by shepherds and veterinary surgeons, who brought the infection to the healthy, and it was also transmitted by dogs and other animals which had touched the diseased with their hair, wings or feathers. I advised that every diseased animal should be killed, for I maintain that, should they be left to slow death, the costs for medicines, veterinary surgeons, attendants, and other means would be very great; and not only this, but their very presence would assist in the diffusion of the contagion. The Sacred College, however, ordained that this measure was too severe, and that remedies should be tried. And, in truth, they were greatly influenced in this decision by the number of people who pretended they had infallible cures for the infection. But the fact is, that in the cattle, as in the human plague, not every one that takes the disease dies of it. Some recover, thanks to nature rather than to the remedies which are resorted to."

The attempts to cure the disease, as Fleming remarks, only resulted in failure and its indefinite extension.

Edicts were proclaimed forbidding the bringing of cattle from the Campagna into the city districts of Rome, under penalty of death to a layman, and of the galleys for life to an ecclesiastic. The sale of hides was interdicted, and the flesh, horns and fat of the animals were ordered to be buried in deep pits and covered with quicklime.

Religious ceremonies were prescribed, and prayers offered by order of the Sacred College, to stay the progress of the plague. The sale of diseased meat was prohibited and meat inspectors were appointed to supervise the markets, and only such pieces of meat as were stamped with the hot iron of the inspector could be sold.

Lancisi said, "It may perhaps be doubtful whether the eating of diseased meat is hurtful; but still it is best to err on the safe side." The last chapter, as Fleming says of this invaluable work, sums up his admirable reflections on the disease.

> "The steps which a wise government should instantly take whenever (which may heaven avert) the pestilence may again appear upon our borders are these: All roads and bye-paths should be carefully guarded, so that no ox or dog be allowed to enter the country. Any animal so entering should be forthwith destroyed and buried. Should the pestilence, however, gain admission, the separation of the sick from the healthy must be enforced by decree. *Indeed, in my opinion, by far the safest course, is instantly to destroy the animal, and with the pole-axe, so that no infected blood may escape on to the ground*, for in attempting to cure the diseased animal the veterinary surgeon may convey the plague to healthy oxen.
>
> "The healthy cattle must be removed from their former pastures which must now be regarded as contaminated.
>
> "The diseased oxen should be kept in stables to which no one is admitted except the veterinary surgeon or the herdsman. The fountains and vessels used by the animals should be frequently cleaned with quicklime. The clothes of the shepherds also should be fumigated. The dead carcase, from which not one hair is to be removed, must be buried in deep pits, and any saliva or secretions which may drop from them on the way to the pit are to be carefully removed. If any cows are infected their milk is instantly to be thrown into a hole in the ground, and the severest punishment should be inflicted on those who disobey this order. The passage of all rustics and dogs should be forbidden. Such are the means and the reasons for employing them which I offer for the purpose both of

> avoiding and of suppressing the plague. For in truth when I reflect upon the difficulties, the expense, the dangers, and the labours incurred in carrying them out, I confess that I know of no means by which the plague may be more easily, more surely, and more expeditiously suppressed, than by instantly knocking on the head every infected animal, and burying it deeply in the earth. Should this plague ever recur hereafter, posterity may study these pages with some satisfaction, and certainly with profit."

In the month of July, 1714, the plague passed from Holland to England, appearing first at Islington, and Mr. Bates, surgeon to George I., was instructed to make investigation into the nature of the disease. He then states, "About the middle of July the distemper appeared at Islington, and thereupon their Excellencies the Lords Justices, having notice of it, were pleased to command that I should examine into the truth of the report of its being contagious; and ordered the Lord Harcourt, then Lord and High Chancellor, to grant such authority as would be proper to make the discovery. Accordingly, Mr. Milner, Mr. Offley, Mr. Richardson and Mr. Ward, four justices of the peace for the County of Middlesex, were appointed to make the necessary examinations." They proceeded to Islington, where Mr. Ratcliff already had lost 120 out of 200, Mr. Rufford 62 out of 72, and Mr. Pullen 38 out of 87. He points out the difficulty they experienced in obtaining information, as the owners were unwilling to confess to having the disease with them, as no one would then buy milk from them. Mr. Ratcliff, however, gave the account, viz., that they first refused food, the next day had huskish coughs, and voided excrements like clay; their heads swelled, and sometimes their bodies. In a day or two more there was a great discharge of mucus matter by the nose, and their breaths smelled offensively; lastly, a severe purging which ended with death. Recommendations were then made that *all the cows* in the possession of owners of infected cattle should be killed and burnt, or *at least* the sick; that the attendants should be rigorously excluded from communication with other persons; that cowkeepers be made to parcel out their cattle in small lots, etc., and now for the first time the principle of compensation was adopted whereby forty shillings was to be paid to every individual for killing and burying a sick cow *that had not been sick above twenty-four hours.* As usual, he states that attempts at stamping out were nullified because, "Nor was there ever wanting one or other who gave them hopes of a cure." He enters into detailed statements relative to dissections which were made, and points out that the gall bladders were larger than usual, and filled with a bile of natural taste and smell, but of a greener colour; also that the mesentric glands were twice or thrice their normal size.

Owing to the persistent efforts of Mr. Bates and the four gentlemen named the disease was eventually stamped out, the entire loss to the cowkeepers being £24,500, for which sum His Majesty was further pleased, on the solicitation of the four gentlemen, to grant a brief, "but the many false reports that were then industriously propagated to lessen the value of these poor men's losses so frustrated that charity, that the entire sum collected was but £6,278 2s. 6d., which amounted to five shillings and threepence in the pound, computing their loss as above at six pounds per cow."

In a recent French publication the adoption and carrying out of these measures are referred to in the following manner:—"The disease having passed into England in 1714, the English Government saw no other means of arresting its course and saving the great number of animals which were menaced by it, than immolating all those that were infected, and following the advice which Lancisi had given to his country. This sacrifice was about 6,000, and the contagion was destroyed in less than three months, while Holland, which obstinately and uselessly sought for remedies to cure it, had the misfortune to endure its ravages for three years. The English are, then, as we perceive, the first people of Europe who have given us an example of such conduct"

It was estimated that between 1711 and 1714 the mortality of cattle in Western Europe amounted to

1,500,000 head. No means of estimating the deaths of sheep and other ruminants though they suffered very extensively.

During 1717 the plague ceased its ravages in Europe as a general epizootic and finished its career in Hungary, Prussia, Silesia and Bavaria.

In 1774 the Rinderpest still prevailed in Holland owing to the attempt to cure it, and that country remained as a constant source of danger to all those that had dealings with it.

In June of that year the Rinderpest was carried to the South of France from Flanders, into which country it had entered the preceding year. Stamping out was resorted to, on the advices of Vicg-d Azyr, a physician, and the Veterinary Professor Bougelat, and by special decree one-third the value of the beasts slaughtered was reimbursed to the owners.

This invasion cost France 150,000 animals, valued at 150,000,000 francs.

In 1781 the Rinderpest appeared at the Isle of Thanet and is believed to have been brought over from Ostend by two sheepskins, which being thrown on the beach were taken up with seaweed and placed on a dung heap. At these a cow smelt and rolled on them. On the twelfth, by order of Council, removal of cattle was prohibited and infected ones destroyed and buried at least four feet deep.

The Rinderpest had been so severe in Holland in the preceding year that 300,000 head perished.

During the year 1811 we have records of the great loss caused by cattle plague in Austria, Salzburg and Silesia, and in 1814 the plague spread itself over Europe, having been introduced into the Rhine Provinces in the previous year by the commissariat and slaughter cattle accompanying the allied armies under Schwarzenberg on their way to invade France.

From the Rhine Provinces it spread into Switzerland, Austria, Silesia, Upper Italy and France, and we have many records and notes by veterinarians dealing with this outbreak, notably a monograph by M.

Tonnerieux, Veterinary Officer at Girois, who lays great stress on the contagious and infectious nature of the disease in regard to the danger to human beings where the flesh is consumed. He states—" It has been asked whether it is prudent to eat the flesh of such animals (cattle) as had the plague? The troops of the enemy (Germans) before they reached our unhappy country ate with impunity the flesh of such of their cattle as were affected with typhus (Cattle Plague).

They did so with impunity in the districts that were most devastated by the disease which they introduced.

Even the sick used this food when they were laid up in the hospital. Our people did not scruple after this to have recourse to it and they did so with impunity. I ate it myself many times and without inconvenience. It was as good as the meat from the inferior joints of cattle.

That this food is not of a good quality, that it might injure weak and sickly persons and that it consequently ought to be excluded from the market is perfectly true, but that it can introduce a tendency to typhus in those who eat it I cannot believe. Naturally many recipes and so called certain cures were much vaunted, one introduced by the Viscount de Busy being as follows: yeast, an ounce, ordinary beer, one quart. A glass of water or a cup of coffee would have done as much good. Others recommended drugs as Tartar Emetic, Nitre, Lily roots, Gentian, Ginger and Quinine as is common in any epidemic the Pharmacopœia was ransacked for a remedy.

Hurtrel Darboval, was of opinion that medical treatment of the disease was to be commended, as many animals would recover with judicious management, but if they were abandoned to nature or badly treated there would be nearly as many deaths as there were sick.

This veterinarian enumerates the symptoms which do not materially differ from those seen and reported on by other authorities.

Professor Gohier, who witnessed the epizootic in the department of the Rhine, stated that he quite believed that goats would contract the malady and says that many have taken through staying but a short time in the same sheds with diseased cows.

This authority advocated the slaughter of affected animals when they had reached the second stage of the disease, because his observations had taught him that recovery was then impossible and by killing them at this time the centres of infection were diminished.

In dealing with so-called remedies and preventive measures (setoning, drenching, etc.) he goes on to state: "When we are told that a certain number of animals have been cured or preserved by some kind of treatment we must not take this expression to the letter because it can never be proved that all these have been or would have been attacked by the epizootic."

After this epidemic in France we next hear of cattle plague in 1825, as commencing to spread from its supposed home towards the North, South and East, reaching St. Petersburg, Hungary, Wallacia and even the Saxony frontier.

In 1829 and 1830, during the war between Russia and Turkey, cattle plague prevailed in Southern Russia, and from thence spread through Bessarabia, Moldavia and to Russia, Saxony and Austria.

It is a fact worth noting that though Rinderpest infested South Russia from 1828-30, the mortality among cattle was great among the cattle indigenous to the place which were affected, but when the imported cattle of the German colonists contracted the plague the mortality was very great.

In 1836, Rinderpest was raging in Dalmatia the disease having been introduced from Bosnia (and in this year Professor Jessen of Dorpat describes a case of Rinderpest in a goat).

From Dalmatia we find Rinderpest spreading into the kingdom of Naples where it destroyed many cattle, thence extending its ravages to Turkey.

In 1841, the cattle plague appeared in Egypt and raged till 1844, and is said to have destroyed 665,000 head of cattle.

In order to prove whether it generated in Egypt or was imported, M. Renault, Inspector-General of the French Veterinary Schools collected evidence and obtained the strongest proof that the disease was introduced by Steppe cattle.

In 1840, the Viceroy of Egypt sent agents into Anatoba and Karamania to purchase cattle, and considerable herds were collected for embarkation in the towns of Adana, Tarsus and Kazauli. But before their embarkation a destructive epizootic disease broke out among them and caused the death of a great number. More died on board and the survivors on landing at Alexandria were in a deplorable condition.

This transportation was carried on for the two following years under the same circumstances. The disease far from becoming milder seemed to increase in intensity after the landing of the cattle at Alexandria and therefore their proprietors hastened to scatter them in the surrounding plains. The animals carried everywhere the germs of the malady and spread it among animals of their species in every place into which they penetrated.

From thence the contagion extended rapidly into Lower Egypt and the Delta and penetrating even into Upper Egypt where it also attacked the buffaloes and caused three-fifths of the horned cattle to perish.

Thus it was proved before embarkation that the disease had shewed itself and from records it was clearly established that some of the cattle had been obtained from Roumelia, Moldavia and the banks of the Danube, where the disease was in existence. The oxen brought to Egypt belonged to the Steppe breed of Southern Russia and as M. Renault says "this particular breed was seen in Egypt" and in a report by M. Ismail, an Egyptian veterinary surgeon, we get a faithful picture of a Steppe ox when he described the animals as seen when landed at Alexandria.

They were of the middle size, and their proportions irregular, their bodies gross and thick, the head strong and large, their muzzles of a slate colour, their napes furnished with strong hair, the back and loins short slightly arched, their horns were long in the majority, their chest short and lank, the members strong, tail hanging low, gross and furnished with long and strong hair, was what is called light mouse and slate colour, never red or bay.

What is very remarkable, is that on the whole length, and in the middle of the dorso lumbar region, there are hairs of a varied length forming a ridge from the shoulder to the tail, some also had similar hair on the belly, on the jaws and the quarters. Ninety per cent. of the entire herd of Egypt caught the contagious fever; that is to say, in three years 665,000 fell a prey to the malady."

In 1842 and '43 the plague raged in South Russia with great virulence, but this outbreak by severe measures was confined to narrow limits.

Mr. Jearnes, British Consul at Odessa, mentions another outbreak in 1844, and after explaining his belief that the original seat of this Siberian Cattle Plague is Bessarabia, goes on to give the following description of the symptoms and course of the disease.

The affected animal loses its appetite and ceases to ruminate, ears are cold and pendant. Later the eyes begin to water and a slimy foam drops from the mouth, the beast is uneasy lying down and getting up. At length, when too weak to rise, it remains upon the ground moaning; the flanks heaving quickly, and with every appearance of suffering death ensues. In 1865 the Rinderpest appeared in England, having been brought by a cargo of cattle sent from the port of Revel in the Baltic.

It passed thence to Holland, with a herd of Dutch oxen, which had been infected by simply passing through the London Cattle Market, then from Holland it entered Belgium. Thanks to the intervention of M. Bouley, France almost completely escaped. Only a few isolated cases were discovered on the frontier of Belgium, and also a curious epizootic which was limited to the Jardin d'acclimatation in Paris, and was traced to have arisen from the importation of two gazelles sent from London.

The Rinderpest cost England 500,000 cattle, and more than £4,000,000 sterling.

The English outbreak drew the attention of the Governments of Western Europe to the danger of the importation of this disease, and certain sanitary regulations were promulgated everywhere which up to the present has sufficed to eliminate the risk of contagion in the course of commerce.

Once later, in 1870-71, the plague entered France with the German Army; it spread widely over half the departments in France. Statistics, though in other respects very incomplete shew a loss of more than 500,000 head valued at £1,000,000.

As a matter of fact the Rinderpest is endemic, so far as Europe is concerned, in the basin of the Caspian Sea and the neighbourhood of the Ural Mountains and the Caucasus.

Moreover, despite the energetic enforcement of sanitary regulations certain districts of central and Western Russia are still constantly infected and these outbreaks constitute a perpetual menace to Austria and the Balkan States.

The losses are only approximately known. In the steppes of Central Russia where cattle are allowed to run wild, the owners are themselves ignorant of the exact number of deaths. The following figures published in official statistics apply exclusively to European Russia.

Animals affected :—

1884	919,550
1885	350,000
1886	286,000
1887	76,000
1888	87,000
1889	19,000
1890	16,000
1891	2,270
1892	59,212

The average mortality among Steppe cattle is estimated at 20—30 % of the sick. The mortality however among European cattle is we know enormous reaching to from 90 % to 95 %.

Although the Rinderpest is effectively guarded against in Europe it ravages all the regions in Asia and Siberia, Asia and Persia are permanently affected.

The sickness has been found in China whence it was carried in 1892 to Japan; it raged up to March 1894, the losses amounting to 9,716 cattle dying or being destroyed (Janson). Tonkin and Indo-China are equally subject to its ravages

A very ancient focus existed in the Dutch Indies; At Java from 1879 to 1883, 223,443 animals succumbed, representing a loss of £1,400,000, in 1889 there were 22,000 animals affected of which 20,000 succumbed. At the same time the researches of Von Eecke lead one to believe that they have in a measure confused the cattle plague with another malady.

Africa has also been formerly invaded by Rinderpest.

It was localized for a long time in lower Egypt and then had entirely disappeared when in 1890 it was again introduced by cattle purchased for the Italian expedition to Massowah, thence the plague was carried to the higher Nile and the Central regions of Africa by Arab caravans. Sir John Kirk writing to *Nature* in 1896 says "That there can be little doubt that the present epidemic, known under the common name of Rinderpest, is the same as that with which we have been familiar in Central Africa for the past six years, commencing so far as we know in Somaliland in 1889, where the disease carried off a large part of the cattle. It passed through Masoiland in the autumn of 1890. It was there that Captain Lugard, then an officer in the Imperial British East African Company, first came in contact with it. The disease was first reported to me as being at Bulawayo on March 3rd, 1896, and its extension southward is fully and ably dealt with by my colleague Mr. Duncan Hutcheon M.R.C.V.S. the Colonial Veterinary

Surgeon in his special report on Rinderpest in South Africa (March 1896, February 1897).

As Nocard and Leclainche state, up to the present date the plague has been almost invariably introduced into Western Europe by the movements of armies, the only exception is in the case of a limited epizootic which raged in South-West France in 1774. This outbreak seems to have been caused by a consignment of cattle sent by the French Hugenot refugees in Holland to their co-religionists in South-West France. This transmission of the pest by means of commercial dealings entirely unknown up to the second half of the nineteenth century, will probably become more serious owing to the extension of international relations and the progress made in means of transit.

Appendix 5.—HORSE SICKNESS.

In my annual reports for 1894 and 1895 I have entered into a detailed description of this disease for which I suggested the name of Œdemamy- cosis.

Inasmuch, however, as wider observation has admitted of my determining the fact that of the endemic diseases of this country there are a number in which the pathological phenomena have a great similarity, I am now of opinion that to the whole class the term Œdemamycosis might suitably be applied. Of such disease the following might be included :

1. Horse Sickness ... Œdemamycosis equi.
2. Heart-water in sheep ,, ovis.
3. ,, ,, ,, goats ,, capri.

These latter two are identical (See section on Heart-water).

4. A disease in cattle (Œdemamycosis bovis) which I have only seen for the first time during the present year. I learn that it is probably the disease known to the Kafirs as Pungu, or Rivierziekte.

These diseases have peculiar features in common.

1. Almost invariably there is a total absence of inflammatory changes
2. An exudation of serum into the pleural and pericardial cavities, and between the lobules of the lungs.
3. An exudation into the pulmonary alveoli, which at death, may, and in the horse and cattle disease is almost invariably ejected from the nose and mouth as a white froth.

With regard to horse sickness, the advent of Rinderpest, the researches into its nature, and the means which have been adopted to arrest its destructive effects, have demanded so much attention, that but little time was available for investigation into other diseases, nevertheless, certain results which have accrued from my work, are in some measure an advance on what has formerly been done and demand some attention.

The Virus—While former experiments have conclusively shown that defibrinated or citrated blood can be mixed with glycerine and kept for a very long period of time (at least a year) without detriment to the virulence of the contagion contained in it, I have found that drying invariably ensures its destruction if the layer of blood which is being dried is of any appreciable thickness.

Incubation Period.—The inoculation of fluid, fresh or preserved blood, usually occasions a rise of temperature on or about the eighth day when a dose of about 2 cubic centimetres is used.

The use of smaller amounts by subcutaneous injection is uncertain. I have injected one cubic centimetre of recent, though preserved blood, and failed to produce the disease, while a second inoculation of the same quantity of the same blood has successfully produced the onset of the malady.

Where the disease has been thus produced, it has *invariably* proved fatal.

Seton Inoculation.—Inoculation by means of a seton soaked in preserved blood has always produced the disease in a most virulent form.

Serous exudation as a Virus.—The use of the serum, found in so great quantity in the pericardial sac, as a virus is uncertain; sometimes it is virulent, but on other occasions I have injected as much as five cubic centimetres without effect; the disease was not produced, and as proved by subsequent blood inoculation (when the disease in a fatal form supervened) no immunity was conferred.

As a result of blood inoculation under the skin of the shoulder or neck, the pulmonary form is invariably produced in which the lung is pale, and the interlobular spaces are distended by the presence of a clear yellow serum. This condition is that which is commonly found in animals dying on the veld at such times as the disease is present in the Colony.

In Mashonaland, however, it is the rule so far as I know—and lungs of horses which have died there, and which have been sent me confirm this—to find the lungs highly engorged with blood.

This condition is due to a very accentuated form of congestion, but is not associated generally with true inflammatory changes. Moreover, in the colonial form the animal sustains a very high temperature, rarely under 106 Fahr. I am informed that in Mashonaland the temperature is not usually so high.

Conversion of the Colonial form into the Mashonaland.—I have found that if blood from an infected animal with high temperature is drawn off asseptically into citrate solution, and kept at a moderate temperature (without the advent of putrefaction) for 16 days that a change occurs in the virus. In my first experiment of the kind I used blood which had been kept for 16 days, and thereafter glycerinated and kept for twenty-four hours before being used. Of this, 5 cubic centimetres was used to inject subcutaneously on the 10th April, 1897. The temperature rose on the 17th at mid-day to 102·1. The same evening it rose to 103 2, and the following morning it was 103·1. It died the following morning, and the temperature at no time was higher than 103·2. A *post-mortem* examination was made at

7 a.m. on the 19th instant, and the following condition was found:—

Trachea.—No fluid present.

Lungs—Engorged with blood and of a bright red colour. Subplueral and interlobular infiltration present, but small in amount.

Heart.—No ante-mortem clots. The endocardium, especially at the point of attachment of the chordæ tendineae, was the seat of numerous extravasations.

Liver.—Of a dirty grey colour. The lobules were mapped out by perilobular infiltration. Some subcapsular petechiæ were present.

Spleen.—Normal in size, but softer in consistence than normal.

Kidneys.—Deeply engorged with blood.

Bladder.—Containing one-third of a pint of dark brownish fluid which contained much albumen blood corpuscles, oxalate of lime and urates.

Intestine.—Deeply inflamed in patches throughout, the glands being enlarged.

The case was very similar to one I examined in 1892, when the animal was ridden hard into Grahamstown, during a prevalence of horse sickness, and died on the following day. In the case just detailed there were conditions which lent suspicion that senticœmia had possibly been present, and accordingly another animal, No 94, was inoculated on the 19th April with 3 c.c. similar blood, and died at mid-day on the 30th. The highest temperature recorded was 104·4 in the evening of the 29th. In this case no intestinal lesions were found, and the general condition was identical with horse sickness excepting for the great congestion in the lung. Later experiments made with other samples of blood similarly treated have given equal results, and lead to the conclusion that some change is produced in the virus whereby its virulence is increased. The most curious fact, however, is seen in the disease produced, which kills in a shorter period of time, and during which the temperature is never so high as in the ordinary type. It might be held that some new infection had gained access to the virus, and that the death was not due to horse sickness but to septic infection. Against this, however, I have to snow that although this virus has been used in much larger doses to inoculate three salted horses, no illness whatever was produced by such inoculation.

Experiments in the direction of producing Immunity.—In my former reports I have alluded to methods which I had already adopted for salting horses.

I. The first method used consisted in adding to preserved blood sulphuric acid in the varying proportions of 1 in 800, 1 in 1,600 and 1 in 3,200.

The animals were first inoculated with the strongest and after eight days with the weaker and still later with the weakest, after which they were inoculated with a small dose containing merely a trace of acid. Of nineteen animals so treated, one developed the sickness 23 days subsequent to the last inoculation, and several others were slightly sick. Subsequently they were sent to Mashonaland, but owing to drought and accident several died on the way, while of the others some died undoubtedly of horse sickness in Mashonaland. Three however reached Tati, and were used in the Matoppa Campaign.

It was therefore evident that this method would not give protection sufficient to ensure safety in Mashonaland.

II.—*Intensive Method.*—In this method an animal was infected with virus and subsequently given from 30 to 60 grains of calomel daily as soon as the temperature began to rise. At death, or preferably immediately preceding the fatal issue, blood was drawn from the jugular vein and preserved as usual. Experiment has shown that such blood is undoubtedly attenuated, as two animals have been salted in this way without further treatment than the simple inoculation of three cubic centimetres subcutaneously. As a rule, however, I have found that it is safer to dose each animal with calomel as soon as the temperature begins to rise, and I have found that chemically pure calomel does not succeed so well as the ordinary commercial form.

Altogether I have salted nine by this method, and of these six were sent to Bechuanaland two years ago and were exposed to one of the most deadly epidemics known in that country in a succession of years. These all resisted horsesickness, and of these I had two returned to me, which have been again tested by injection of 5 c.c. of virulent fresh blood, and eight days later with 5 c.c. No elevation of temperature occurred, and it was evident that the immunity was still of a high order.

Taking advantage of the fact that a very large amount of serum is found in the pericardial sac after death, I passed several litres through a Pasteur Filter, and injected one horse with 100 cubic centimetres. No rise of temperature followed, and after fourteen days had elapsed I inoculated it with 3 c.c. of virulent blood which had been preserved for several months. As a result the horse developed typical horsesickness and died. There was no indication of any, even the slightest degree of immunity having been produced.

The Serum of Immune or Salted Animals.—One old animal which I had salted two years previously was re-inoculated with 5 c.c. of virulent preserved blood. After twelve days had elapsed, during which no rise of temperature had occurred, the animal was bled and serum obtained from the blood.

My object in the first instance was to determine the value of serum of blood of animals already salted, for if the blood of salted animals to which only a small dose of preserved virus had been given could immunise, then the possibility of farmers making use of salted horses in this way would manifestly be of great practical importance.

An experiment was then carried out with the serum derived as stated.

Horse No. 95 was inoculated with 100 cubic centimetres of the serum and immediately afterwards with five cubic centimetres of preserved virus.

No. 96 was inoculated with an equal amount of virus as control. Nine days subsequent to inoculation the temperature began to rise in each, and on the following day it was higher in the one that had received the virus and serum than in the one to which no serum had been given. On the 11th day, No. 95 was again inoculated with 10 c.c. serum, but without any appreciable effect. It died on the 12th day, and the control died on the day following.

Horse 79 was then inoculated with 10 c.c. serum and immediately afterwards with 2 c c. preserved virus. These inoculations were made on September 3rd. On September 7th, the temperature being still normal, another 100 c.c. was injected. On the 11th the temperature began to rise, and another 100 c.c. was given. On the 12th, 13th and 14th, 100 c c. was injected each day, the temperature having risen steadily each day until the 14th when it fell. On the 15th no serum was given, as the temperature was low, but as it rose in the afternoon, a dose was given early the following morning. It died in the forenoon of the 16th.

No. 101 was inoculated with 2·5 c.c. virus on the 18th September and 100 c.c. serum on the 21st, and each day subsequently till October 1st it received 100 c.c., making in all 1,200 c.c. of serum which it had received. It died of horsesickness on the 1st October.

It seemed evident, therefore, that the serum of the blood of a salted animal was of very little avail in arresting the progress of the fever of horse-sickness.

I now determined upon testing the value of serum from salted horses which subsequent to "salting" had been inoculated with progressively increasing doses of virulent blood. I accordingly arranged to have two horses which, having been "salted," had been exposed in the veld at Bechuanaland for nearly two years. These two horses ("Folly" and "Charlie") were accordingly sent to the Institute and arrived on October 31st.

On the following day each was inoculated with 5 cubic centimetres of virulent preserved blood. On the 9th each received 50 c.c. fresh virulent (unpreserved) virus. On the 20th each received 200 c.c. fresh virus; on December 30th each received 500 c.c. fresh very virulent virus. On the 19th, Folly had a slight rise of temperature in the evening; on the 20th, Charlie had a slight rise. These rises were evanescent and may not have any relation with the effect of inoculation. On the 30th December each received 500 c.c fresh virulent blood. No reaction occurred. On November 29th, Charlie was bled and serum obtained from the blood.

On December 1st, No. 110 received 2 c.c. virulent blood (preserved), and immediately thereafter 50 c.c. of this presumably fortified serum.

No. 111 was treated in exactly the same manner, and the subsequent injections of serum may best be shewn in a tabular form.

	No. 110.	No. 111.
Dec. 1 ..	2 c.c. virus & 50 c.c. serum.	2 c.c. virus & 50 c.c. serum.
,, 2		
,, 3 ..	.. 50 ,, ,,	
,, 4 ..	..	.. 50 ,, ,,
,, 5 ..	.. 50 ,, ,,	
,, 6		
,, 7 ..	.. 50 ,, ,,	.. 50 ,, ,,
,, 8		
,, 9 ..	.. 50 ,, ,,	
,, 10 ..	..	.. 50 ,, ,,
,, 11 ..	.. 50 ,, ,,	
,, 12		
,, 13		
,, 14		
,, 15		
,, 16		
,, 17 ..	.. 200 ,, ,,	
,, 18 ..	.. 100 ,, ,,	
,, 19 ..	.. 200 ,, ,,	
,, 20 ..	.. 200 ,, ,,	
,, 21 ..	.. 200 ,, ,,	21st, died.
,, 22		
,, 23 ..	died.	

In both cases on the 12th day after the first inoculation and as no rise had yet occurred each animal was inoculated with 5 c.c. of virulent blood but as on the second day following the temperature began to rise it is evident that it had been due to first inoculation which had been delayed by the giving of the serum.

I therefore determined on a fresh experiment in which serum should be sparingly given until such time as a rise of temperature should occur.

Accordingly Nos. 113 and 114 were each inoculated on the 22nd December with 2 c.c. virus and immediately thereafter in a different situation with 50 c.c. serum Five days later each received 100 c.c. serum. On the same evening the temperature began to rise and on the subsequent day each received 100 c.c. in the morning and 200 c.c. in the evening. Next day no serum was given as temperature was remaining stationary. Curiously the symptoms now shewn were very different to those usually seen and the urine was highly charged with blood. On the following day a dose of 100 c c. was given to each. On the 31st inst. 200 c.c. was given to each in the morning subcutaneously, as usual, and in the evening 100 c.c. was injected into the jugular vein of each animal. On the following morning the temperature of each animal was falling. No. 114 died the following evening and in the case of No 113 a slight rise again occured on the 3rd January when it was deemed advisable to give 100 c.c. of serum. The temperature now fell and it died on the evening of the fifth inst. The post-mortem examinations in this case revealed no characteristic lesions of horse sickness.

The subcutaneous tissue was of a brilliant yellow colour; the liver was congested and bile stained. The kidneys were large and pale, and the bladder filled with a deeply blood-stained urine. The blood was very peculiar in appearance, being of a pale colour, watery in density and uncoaguable. The veins contained large post-mortem clots which were quite translucent in appearance.

At this time another animal uninoculated developed a high temperature and began to pass bloody urine. All treatment was unavailing and it died in collapse. The post-mortem examination shewed similar conditions to the two former cases and with regard to the detailed examination I hope to give these at a later period Meanwhile there is no doubt that the uninoculated animal suffered from an acute specific disease which in its history both before and after death had a resemblance to Texas Fever of cattle and its appearance in the two inoculated animals most probably occasioned their deaths which were not primarily due to horse sickness inasmuch as the post-mortem examination failed to evince any of the phenomena which are associated with that disease.

After having had the stables entirely cleaned and disinfected a new set of experiments were conducted in which a dose of serum was given at least twenty-four hours previous to the inoculation of virus. After several days a further dose of serum was given and, as soon as the temperature began to rise, daily doses were given until the animal recovered.

The following records shew the result of inoculation in two animals, one being treated with serum and virus, the other with virus alone.

No. 119		
Jan. 24	100·3	50 c.c.
	100·4	serum
25	101·3	
	101·2	2 cc.
	101·2	
	101·0	virus
	102 3	
26	101·1	
	100·4	
	100·3	
	101·0	
	102·0	
27	100·2	
	101·3	
	101·2	
	99·2	
	101·2	
28	100·1	
	101·0	
	99·4	

No. 119.		
Feb. 3	101·2	50 cc.
	100·4	serum
	102·0	
	102·0	100 cc.
	104·0	serum
4	103·1	100 cc.
	103·4	serum
	104·2	
	105·0	100 cc.
	105·0	serum
5	102·1	100 cc.
	104·0	serum
	106·1	
	102·0	
	104·0	
6	103·2	100 cc.
	104·0	serum
	104·1	
	103·1	
	102·4	

No. 120.		
Jan. 26	100·0	
	100·0	5cc. virus
	100·2	
27	99·0	
	100·1	
	99·4	
	100·0	
	101·0	
28	98·1	
	99·2	
	99·2	
	100·2	
	101·0	
29	99·4	
	99·3	
	100·0	
	101·1	
	101·1	
30	99·4	
	100·0	

No. 119.

28 101·0
101·0

29 100·1
100·1
99·2
100·4
100·3

30 99·2 } 100 cc.
100·1 }
100·4 serum
102·0
103·2

31 99·2
101·1 100 cc.
102·0 serum
102·3
104·3

Feb. 1 101·2
101·2 100 cc.
102·0 serum
102·0
103·2

2 101·2 100 cc.
102 1 serum
102·0
102·3 50 cc.
104·2 serum

No. 119.

7 102·0
102·1
Recovered.

No. 120.

99·1
101·0
101.3

31 100·3
100·3
101.0
102·0
102·3

Feb. 1 101·2
102·0
102·3
103·3
102.3

2 101·2
102·0
102·3
103·3
104·3

3 103·3
103·3
104·0
105 0
104·3

4 103·2
103·0
103·2
Died virulent
Horse-sickness.

In these experiments it will be seen that by the use of serum a curative action can be induced, but a very considerable amount of serum is required. In this case about one litre of serum was used in order to effect a cure. I am now engaged in the attempt to produce such a satisfactory result with an expenditure of less serum, for the employment of such a large quantity would be against the practicability of this method on a large scale.

It is a matter worthy of note that the virus used in this case was preserved and had been kept for more than a year, and should the serum method come into general use, the fact that virulent virus can be supplied to the farmers will be of great utility, as a certain and sure infecting agent can then be relied upon.

The virus of horse-sickness is most variable in effectiveness, and since a considerable quantity can be obtained from one animal, it will be possible to test batches of serum in relation to and against a specific batch of virus.

I consider, therefore, that the serum properly prepared may be looked upon hopefully in the absence of any other method, as calculated to serve for the "salting" of horse against horse-sickness.

APPENDIX 6.—HEART WATER.

This peculiar disease has devastated many parts of the Eastern Province of sheep. Indeed its prevalence is so well marked that sheep breeding which was at one time a large and lucrative industry in the Eastern Province has been almost lost.

A disease having nearly if not indeed identical symptoms also obtains among imported strains of goats and the object of my enquiry has been devoted in the first instance to the question whether the disease affecting sheep and goats is identical in both cases, secondly whether it is due to some fault in or deterioration of the pasture lands, or is due to some infective agent, and thirdly, if it is an infective disease, whether or not there is any prospect of devising a method of protective inoculation.

According to former accounts, especially those of Mr. Soga, Assistant Veterinary Surgeon, the animals die very soon after being noticed to be ill and on post-mortem examination a considerable quantity of fluid is found in the chest. He made several experiments with the object of attempting to infect healthy stock but did not succeed in so doing.

During the past two years I have made from time to time as circumstances and occasion permitted some preliminary experiments which led me to conclude that the disease was truly an infective one, was identical in sheep and goats and was communicable from one to the other. For the purpose of carrying out such experiments I have endeavoured when I could to obtain suitable cases for conveyance to the Institute as circumstances there admit of exact experiments being conducted with greater exactitude than in the open veld.

On the 16th December, Mr. George Webster of Middleton wrote to me that goats were dying and begged me to make an enquiry into it. From his description I was convinced that the disease was Heart Water and I therefore wrote him, asking him if he could possibly obtain a case for me in the early stage and if so it might be arranged to convey it here. Mr. Webster kindly obtained from Mr. Turner a good case in the primary stage and we had it brought to the Institute. On the 12th January the goat arrived here late in the evening. On arrival, there was very little to be noticed as a departure from health. It was slightly dull and listless and had a high temperature The latter peculiarity was not however to be considered of serious moment because goats when moved about frequently develope a very high temperature though in full health. Fearing however that acute symptoms might develope during the night I drew a little blood from the jugular vein which was then placed in the ice chest in order to be used for the inoculation of some healthy animals on the following day. On the day following the goat was distinctly seedy and had a temperature, even when housed in a stable, of 106 Fahr. Towards evening its breathing was somewhat accelerated. In the morning the breathing being more marked and slight convulsive symptoms being evident as a near approach of death a further small quantity of blood was drawn from it. It died very shortly afterwards, and a post-mortem examination was then made

As regards the pathology of this disease, I can at present speak only in general terms, leaving the more exact details for another report.

The conditions seen on such examination were as follows :—

The trachea contained some white froth and this was also present in the bronchi and smaller lung tubes. Some slight exudation seemed to be present under the pleural membrane reflected upon the lung surface.

The pleural cavity contained a large quantity of pale yellow serous fluid among which some flocculi were apparent.

The pericardial cavity was filled with serous fluid which was faintly blood stained.

Some slight changes were visible in the contents of the abdominal cavity, principally in relation to the stomach of which the walls were slightly odematous and the mucus lining was readily separable. From the outside the appearance of the rumen was as if it were filled with soft clay or putty. The blood was readily coagulable.

The gall bladder was tense and filled with bile, its outer surface being of a dark grass green colour. On withdrawing the bile its colour was of a beautiful olive green, clear and limpid, with slight mucous coagula suspended in it.

With some defibrinated blood on the 14th January I inoculated one merino goat No. 1 and one sheep No. 1, each with 10 c.c. subcutaneously and 10 c.c. intravenously, into the lumen of the external jugular vein.

On the following day I inoculated one merino goat No. 2 and one sheep No. 2 subcutaneously with 10 c.c. of the bile.

In the case of the blood inoculated animals, the goat had a sharp rise of temperature eight days after inoculation after which it descended on the following day, had an irregular temperature during four days followed by four days of high temperature (about 107 Fahr.) and died on the morning of the 1st February.

The sheep inoculated with blood had a sudden rise of temperature on the fifth day after inoculation which immediately thereafter fell to normal and was followed by a gradually rising temperature.

Both the goat and sheep shewed peculiar convulsive movements on the morning of the 1st February and died within two hours of each other. At the post-mortem examination in both cases, the characteristic symptoms of Heart Water were found, the pleuræ and pericardial cavities contained enormous quantities of yellow serous fluid and of which the pericardial was slightly blood stained.

The pericardium in the sheep was actually ruptured owing to the tension of the fluid within.

The bile inoculated animals each shewed a sharp rise of temperature about one week after inoculation which was succeeded by irregular high temperature for some days and then descended to normal.

The character of the temperature compared with that of the blood inoculated animals suggests in a most pronounced manner that the animals inoculated with the bile have each suffered from the disease in a mild form.

In order to determine this point the animals have been inoculated subcutaneously and intravenously with the blood of the animals that have died and control animals have at the same time been inoculated with the same blood.

The result will be communicated at a later date.

The experiments so far shew that Heart Water is due to an infective principle which can be communicated from animal to animal of each species, namely from sheep to sheep and from species to species namely from goat to sheep.

Should it be found that the bile actually conveys the disease in a mild form, it will be requisite to determine the smallest quantity which will act satisfactorily because the gall bladder only contains about 50 c.c.

Also seeing that it is not practicable for the farmer to house the sheep or goats, inoculation to be successful would require to be performed during the season when the disease does not exist in the veld.

APPENDIX 7.

RED WATER OR TEXAS FEVER.

The disease known as Red Water in South Africa has received this familiar appelation from the fact which has been noticed by the farmer that the urine voided by the animal is of a red colour.

The name of Texas Cattle Fever was applied to a disease which affected the cattle of Northern States after they had come in contact with cattle from Texas or Southern States. A peculiar fact which made the onset and cause of this disease most mysterious was that the Southern cattle, from which the clean cattle from the Northern States were supposed to be infected, were themselves perfectly healthy.

It was further noticed that these Southern cattle were not directly infectious, but that the ground over which they passed was infected by them, and that from this ground infection passed to healthy susceptible cattle which passed over it.

Further, it was found that the Southern cattle, after having been in Northern lands for some little time, lost their power of infecting other cattle.

American cattle owners also were unanimous in their belief that native susceptible cattle which became infected with the disease did not transmit it to healthy natives.

In this country we have learned by experience that Red Water cattle can and do infect the veld.

When this disease first made its appearance an enormous mortality followed, but now we know that cattle born in so-called "Red Water velds" are themselves proof against Red Water.

I had no intention this year of reporting on this subject because I felt it would be necessary to enter in great detail into the pathology of the disease. Time does not admit of this being done now, and I have only dealt with the subject owing to the fact of certain grave results coming under my notice following inoculation for Rinderpest in which Red Water was produced as a complication.

I may state in passing, that American experts made a study of this disease extending over seven years, and that they found a peculiar parasite to be commonly present in the red corpuscles of the blood, and that from the constant presence of this parasite in the blood of animals suffering from the disease they concluded that it was the essential cause of the malady.

They further believed that the carrier and transmitter of the parasite was the cattle tick *Boophilus bovis* (Riley)

Without entering into the details as to the pathology of this disease, I may state that Mr. Hutcheon believed it to be identical with the Red Water of this country. The discovery of a similar parasite in the blood of cattle in the native reserve at Taungs serves to further confirm this view.

In my report I have referred to this, and the fact which seemed so peculiar was that although I found it in some instances in the blood of animals infected with Rinderpest, the numbers of corpuscles which shewed parasites within them were very few in number, and no symptoms of Red Water was associated with it during life.

At that time I reasoned that as the area had formerly been one of active infection, that possibly most of the cattle had become immunized, and that the effect of Rinderpest had been to partially overturn the immunity and so admit of the infection of the blood corpuscles.

Since coming south to the Eastern Province inoculation for Rinderpest has progressed with great rapidity, and that inoculation has involved the use of the blood of animals suffering from Rinderpest for the injection subcutaneously into others. In one or two areas the mortality which followed was comparatively high, and the animals in numerous cases voided a red-coloured urine.

It seemed to me that Red Water had actually been produced, yet in certain cases which I was able to follow up the Rinderpest animals from which the blood had been taken had shewn no evidence whatever of their having any such disease.

In my investigation the following experiment was made:—A black heifer from a well established Red Water area was infected with clean Rinderpest blood from a known strain, namely, that which I had at the Institute.

After she had developed Rinderpest the blood was carefully examined. Two clean cattle at the Waai Nek camp and two in the Institute were inoculated with her blood. These cattle were obtained from an area known to have been up till now absolutely free from Red Water, hence I judged that the four animals were susceptible to that disease.

In all four animals the temperature rose as if Rinderpest only was acting, until about the seventh day when slight muscular tremors and swaying of the hind quarters, with an emission of port wine coloured urine, suggested the presence of Red Water, which was, however, fully confirmed on microscopical examination of the blood in the red corpuscles of which the parasites were easily and clearly distinguished.

From the blood of these two a small heifer and a small ox, which were fully immunized to Rinderpest, were inoculated.

I could not ascertain definitely the history of these two salted animals, but one in particular, the ox, was believed to come from an area that had been free from Red Water.

Both were inoculated on 26th November, 1897, each receiving 5 c.c. of blood subcutaneously and 5 c.c. into the jugular vein.

The temperature of both animals (Nos. 3 and 4) became slightly irregular and rather higher than normal after six days, although seldom over 105 during the hottest period of the day.

On the 11th December, or sixteen days after inoculation, no parasites having been discovered in the blood, some blood was withdrawn from the jugular vein of each animal, and two new clean cattle, Nos. 5 and 6, were respectively inoculated from each, No. 5 being inoculated from No. 3, and No. 6 from No. 4. Each received 5 c.c. subcutaneously and 5 c.c. in the jugular vein.

No. 6 had a rise of temperature on the 15th December or four days after inoculation, and in view of this possibly being due to Rinderpest I gave the animal a dose of Rinderpest serum. The temperature still gradually rose, and on the 19th December examination of the blood revealed the presence of numerous parasites, which, however, were slightly altered from the typical form, many being in the form of small globular bodies in the corpuscles and shewing intermediate forms between this and the paired American form as described by Dr. Smith. The animal died on the 30th December. No Red Water appeared either during life nor in the bladder after death, but all other symptoms except those of the kidney were readily discerned.

From this animal a series of Rinderpest salted cattle were inoculated, none of which died, although in the case of one of them (No. 19) the blood became so thin and watery as to be translucent when viewed through a volume of blood three and a half inches thick. This blood would not coagulate, and although whipped with a wire egg-beater for thirty minutes no fibrin separated. The animal fed all the time, never showed any symptoms other than weakness and has since quite regained health.

My object in thus injecting these Rinderpest salted cattle with Red Water was that, should Rinderpest-Red Water spread we might possess a serum from animals immunised to both diseases with which to inoculate sick cattle.

These experiments abundantly prove that the blood of an animal from a Red Water area, which in itself gives no indication of Red Water, may yet transmit the disease in a virulent form to any susceptible animal.

In the case of the last series of animals the typical paired pear-shaped parasites never appeared, and where parasites were seen they appeared as globular bodies, usually within the red blood corpuscles and in some few instances in a free state. The appearance of this form of the parasite conforms to that described by Drs. Turner and Kolle as appearing in the blood of some animals at Kimberley.

The blood of the animals in which this form of parasite appeared was excessively watery in appearance, the corpuscles were irregular in size, and coagulation either did not occur in the drawn blood, or at best only caused it to become thicker than freshly drawn blood. Inoculation of this blood into clean cattle drawn from Red Water areas caused only a slight irregularity of temperature.

The blood of these animals was also caused to become much thinner than normal, but I did not observe any parasites in the corpuscles.

This matter will still remain the subject of experiment, inasmuch as the experiments made suggest the probability that by inoculating virulent Red Water blood into partially immune animals the virulence of the contagium becomes by successive transfers greatly attenuated.

Appendix 8.—LOCUST FUNGUS.

Six years ago the Secretary for Agriculture directed my attention to the enormous depredations which were being caused by locusts, and asked if I could suggest or attempt any method for their destruction.

From my experience with disease producing fungi among salmon and trout in the rivers and lakes of Scotland (*vide* my Report to Her Majesty's Fishery Board for Scotland, 1890), and my observations on insect destroying fungi, I felt convinced that in this country, where the conditions for the growth of fungi are existant in a high degree, we should find sooner or later that locusts would be attacked by some low form of vegetable life, and I accordingly directed the attention of the

Secretary for Agriculture to such a probability, and asked that instructions should be sent out to all districts asking that wherever locusts were found to be dying, samples should be forwarded to me, and that if I found any depredatory fungus I would attempt its cultivation in a state of purity so as to admit of its dissemination in areas visited by locusts, provided that such fungus should be proved to be harmless to farm stock.

On the 14th March, 1896, I received a telegram from the Under Secretary for Agriculture, asking if arrangements could be made to assist the Government of Natal in regard to the isolation and propagation of a fungus which was destroying locusts in that country. I replied that I should be glad to give all the assistance in my power, the more so since I had been in anticipation that such a fungus would be found sooner or later. The facts relative to its discovery are these: a farmer near Richmond observed that locusts were dying in great numbers, and having obtained some of these he took them to Mr. Arnold Cooper, a gentleman who takes a lively scientific interest in all departments of microscopy. Mr. Cooper recognized the presence of a fungus, and having directed the attention of the Government of Natal to the discovery, suggested the advisability of procuring our assistance. Their request having been warmly acceded to, Mr. Cooper came to the Institute here, bringing with him numbers of infected locusts.

Owing to my having to leave for Cape Town on urgent official matters, I instructed my former assistant, Dr. Black, to give every attention to the matter and to afford Mr. Cooper every facility for any experiments he might wish to carry out, These gentlemen found that it was possible to transmit the fungus growth to artificial culture media and eventually to procure on such media a pure culture of the fungus The determination of the presence of a fungus in the first instance by Mr. Cooper reflects very great credit on that gentleman, and the fact of his being a member of the legal profession, who, as an amateur, gave up most of his valuable time to a matter of such importance to the agricultural community, is a matter deserving of the highest commendation. The work and experiments made by Dr. Black were extremely satisfactory and enabled us in the first instance to prepare the fungus in such a manner, as regards purity and quantity, as admitted of its being made use of on a considerable scale in the field. Since then I have endeavoured to find on what medium it can grow with greatest celerity, and in what manner it can be transmitted to great distances for use in the field. So far I have preferred the use of a saccharine agar whose reaction is faintly acid. Small tubes containing such medium are inoculated, plugged with cotton wool and incubated up to the moment at which visible growth is to be recognised by the naked eye. As soon as this occurs, the plugged end is immersed in liquified hard paraffine, and when cooled such tubes are ready for transmission to distant parts.

For the purpose of infecting an area it suffices to break such a tube on the ground either in high grass or in a suitable shade. It is, however, of the first importance to have a certain amount of moisture present, and hence its use is always most likely to be successful after rain. It is not advisable to use it during a drought unless the place where it is deposited is kept damp.

Various persons who have used it prefer to break a tube in water and to place some living locusts therein, which are subsequently released among a swarm. In Bulawayo the authorities are highly pleased with the success which has attended its use, but they insist on the greater advantage which follows if it is used during the "voetganger" stage.

Quantities of the Fungus have been sent to various parts of South Africa, to North America, the United States, India, Australia and to Buenos Ayres.

In various parts where it has been used most successful results have followed.

I regret that owing to the urgent nature of the work in other departments time has not admitted of the full study of its life history which I am desirous of having made, the more especially since its growth on artificial media gives such results as render the question of a symbiosis, as occuring in its life history, deserving of some attention. It is, however, probable that the fungus is the Empusa grylli of the Entomophthorea, a group of Fungi which for the most part live on or in the bodies of insects into the cavities of the bodies of which they usually penetrate during life. After penetrating into the body cavity they develope hyphæ or filaments on branches of which are borne gonidiophores, *e.g.*, carriers of brood calls. These hyphœ penetrate from within outwards the body of the insect after death, and up on the outer surface the final stages of development are completed. The Entomophthoreœ do not live exclusively on insects. One species, according to the observation which have been made by Lietget, exists as a parasitic fungus in the cells of the prothallia of ferns.

Quite recently it had been reported that a different fungus from the Empusa found in South Africa has been recognized as attacking locusts in Buenos Ayres, and I am now endeavouring to procure some of the infected locusts from that country. It seems to me that only by the maintenance of a warfare against them carried out by the dissemination of parasites, either vegetable or animal, can we hope to overcome them.

Appendix 9.—TABLES OF TEMPERATURES.

(I.)—Inoculations with virus which had been made to undergo physical change by additions of chemical agents.

No. 1.			No. 2.		
October	16		October	16	
,,	17	101·4	,,	17	101·4
,,	18	101·4	,,	18	102·6
		103·6			102·8
,,	19	101·0	,,	19	101·0
		102·0			102·0
		102·6			102·2
,,	20	101·6	,,	20	101·0
		102·0			101·6
		102·6			103·0

No. 1.

October	16	
,,	21	101·6
		102·4
		102·4
,,	22	101·6
		102·0
		102·4
,,	23	101·2
		101·6

No. 2.

October	16	
,,	21	101·4
		101·6
		102·0
,,	22	101·6
		101·6
		102·2
,,	23	101·0
		101·6

No. 3.

October	16	
,,	17	101·4
,,	18	102·2
		102·2
,,	19	101·2
		102·0
		102·0
,,	20	101·3
		101·2
		102·0
,,	21	102·0
		102·0
		102·0
,,	22	101·0
		101·3
		102·1
,,	23	101·4
		102·2
		102·3
October	24	102·0
		103·3
		103·4
,,	25	100·3
		102·0
		102·1
,,	26	100·3
		102·0
		102·3
,,	27	102·3
		101·2
		101·3
,,	28	101·1
		102·3
		102·0
,,	29	103·2
		101·2

No. 4.

October	17	
,,	18	101·4
		104·0
,,	19	101·4
		104·0
		103·0
,,	20	102·0
		102·6
		103·6
,,	21	101·6
		102·0
		102·4
,,	22	101·0
		102·0
		103·2
October	23	102·6
		102·0
		103·0
,,	24	100·6
		101·6
		102·6
,,	25	100·6
		102·2
		102·4
,,	26	100·8
		102·0
		103·8

No. 5.

October 18	..	101·2	October 24	..	101·2
		102·6			101·6
,, 19	..	101·0			104·0
		102·0	,, 25	..	100·6
		102·4			101·6
,, 20	..	101·6			102·2
		102·0	,, 26	..	100·6
		103·0			102·6
,, 21	..	101·0			102·8
		101·6	,, 27	..	102·2
		102·0			102·2
,, 22	..	101·4			102·6
		102·0	,, 28	..	101·0
		102·6			102·0
,, 23	..	102·0			100·0
		101·8			
		101·8			

(II.)—Inoculation with old virus producing modified attack of Rinderpest.

No. 6.

October 20	..	102·6	October 25	..	105·0
,, 21	..	102·0			106·2
		102·4			106·8
		103·0	,, 26	..	103·6
,, 22	..	104·0			104·4
		106·4			104·6
		107·4	,, 27	..	102·4
,, 23	..	102·4			102·6
		103·4			103·6
		105·0	,, 28	..	101·8
,, 24	..	105·2			103·2
		107·4			102·8
		107·8	,, 29	..	101·2

Inoculated with 2·5 c.c. of citrated blood which was kept for seven days aseptically before being used.

The result in this case was to produce a modified attack with recovery.

Subsequent inoculation with virulent fresh mucus by smearing the nose was followed by a second attack from which the animal eventually recovered.

(III.)—Animals inoculated with blood from Rinderpest affected animal, which had been kept nine days at ordinary temperature, Rinderpest was not produced.

No. 7.		No. 8.	
October 23 ..	101·2	October 23 ..	101·8
,, 24 ..	101·8	,, 24 ..	100·6
	102·6		102·6
	102·6		104·0
,, 25 ..	101·0	,, 25 ..	101·6
	102·6		102·4
	101·2		103·2
,, 26 ..	101·6	,, 26 ..	101·0
	103·0		104·2
	103·8		103·2
,, 27 ..	100·6	,, 27 ..	101·0
	103·2		102·6
	103·4		101·4
,, 28 ..	101·4	,, 28 ..	103·2
	102·0		103·8
	101·8		102·6
,, 29 ..	101·4	,, 29 ..	100·4
	102·6		102·2
	102·0		100·4
,, 30 ..	100·0	,, 30 .	100·0
	102·4		102·4
	102·6		102·6
,, 31 ..	100·4	,, 31 ..	100·4
	102·0		101·6
	102·4		102·6
November 1 ..	101·0	November 1 ..	100·0
	102·6		103·0
	103·0		103·0
,, 2 ..	100·0	,, 2 ..	100·0
	102·4		102·4
	102·6		103·0
,, 3 ..	104·0	,, 3 ..	100·6
	102·2		103·0
	102·6		102·0

(IV.)—Animals Inoculated with Virulent Blood.

No. 9, with 2 c.c.

October 23	Inoculated	October 26 ..	103·8
,, 24 ..	101·2		105·4
	102·4		106·2
	102·0	,, 27 ..	103·8
,, 25 ..	102·0		105·6
	102·6		106·2
	103·6		

October 28 ..	101·6
	105·4
	103·0
,, 29 ..	103·8
	104·2
	105·0
,, 30 ..	103·0
	105·4
	105·4
,, 31 ..	103·8
	104·6
	103·0
Nov. 1 ..	103·2
	106·2
	105·4
,, 2 ..	105·0
	106·6
	105·6
,, 3 ..	104·0
	104·8

Death at mid-day.

No. 10,

with 2·5 c.c.

Oct. 23	Inoculated
,, 24 ..	101·2
	101·4
	103·0
,, 25 ..	100·0
	102·0
	102·2
,, 26 ..	101·2
	102·0
	104·6
,, 27 ..	104·6
	107·0
	107·0
,, 28 ..	103·2
	105·0
	105·6
,, 29 ..	104·2
	107·0
	105·0
,, 30 ..	104·0
	107·2
	103·2
,, 31 ..	103·2
	104·2
	106·8
Nov. 1 ..	102·6
	107·6

Died.

No. 11,

with 2·5 c.c.

Oct. 23	Inoculated
,, 24 ..	100·2
	101·6
	102·0
,, 25 ..	101·0
	101·6
	102·8
,, 26 ..	101·0
	102·6
	103·8
,, 27 ..	103·6
	105·0
	105·8
,, 28 ..	103·6
	105·6
	104·2
,, 29 ..	105·0
	106·0
	106·0
,, 30 ..	104·8
	105·8
	106·4
,, 31 ..	104·2
	106·2
	103·4
Nov. 1 ..	104·0
	105·4
	106·0
,, 2 ..	100·0

Died.

No. 12,

with 2·5 c.c.

Oct. 23	Inoculated
,, 24 ..	100·2
	102·6
	103·0
,, 25 ..	100·0
	102·2
	103·2
,, 26 ..	101·6
	102·8
	102·6
,, 27 ..	103·6
	105·0
	105·6
,, 28 ..	101·4
	105·4
	103·8
,, 29 ..	102·2
	103·8
	106·0
,, 30 ..	103·0
	106·6
	106·4
,, 31 ..	104·4
	107·0
	106·8
Nov. 1 ..	104·8
	107·0
	106·6
,, 2 ..	105·0
	106·8
	105·4
,, 3 ..	100·0
	100·0

Died.

(V.)—Animals inoculated with citrated Rinderpest blood to which glycerine was previously added.

No. 13.		No. 14.			
Oct. 25 ..	101·8	Oct. 25 ..	101·8	Nov. 4 ..	101·4
	102·4		101·4		104·8
	102·0		102·6		102·2
,, 26 ..	101·4	,, 26 ..	101·4	,, 5 ..	103·0
	102·0		102·0		103·8
	103·8		103·6		105·4
,, 27 ..	102·2	,, 27 ..	103·6	,, 6 ..	104·6
	103·0		103·0		100·2
	103·0		104·8		106·6
,, 28 ..	104·6	,, 28 ..	101·4	,, 7 ..	103·8
	102·6		104·8		105·0
	103·2		103·2		105·8
,, 29 ..	101·6	,, 29 ..	105·2	,, 8 ..	103·0
	102·0		106·6		105·0
	105·4		106·4		105·0
,, 30 ..	104·6	,, 30 ..	100·4	,, 9 ..	102·4
	107·4		101·4		106·8
	106·0		102·6		105·4
,, 31 ..	105·0	,, 31 ..	100·4	,, 10 ..	100·4
	106·2		100·6		105·0
	107·0		103·8		104·6
Nov. 1 ..	103·2	Nov. 1 ..	100·0	Died.	
	107·4		102·0		
	106·0		102.6		
,, 2 ..	105·0	,, 2 ..	100·0		
	105·0		101·2		
	105.0		102·1		
,, 3 ..	102·0	,, 3 ..	100·4		
	104·4		102·0		
Died.			104·0		

No. 35 was inoculated by sponge saturated with nasal mucus from a virulent case at Schaapfontein (Phokwani). The mucus when used was twenty-four hours old.

January	14	..	101·2
			100·2
,,	15	..	101·1
			102 0
			101·3
,,	16	..	101·0
			104·2
			104·0
,,	17	..	103·4
			103·0
			101·1
,,	18	..	101·4
			100·3
			100·2
,,	19	..	101·2
			101·0
			102·0
,,	20	..	103·1
			103·0
			106·4
January	21	..	103·0
			103·3
			104·2
,,	22	..	103·1
			104·3
			104·4
,,	23	..	104·4
			104·4
			103·1
,,	24	..	104·0
			104·2
			104·0
,,	25	..	103·4
			104·0
			104·2
,,	26	..	Recovery.

Temperature now became normal and remained so.

Dry sawdust, previously sterilized, was moistened with virulent Rinderpest blood and dried at room temperature for seven days. Each animal was given two grammes by the mouth.

No. 192.

November	18	102·3
		103·0
,,	19	100·2
		100·4
		102·3
,,	20	100·2
		102.0
		102·3
,,	21	100·0
		103·0
		102·3
,,	22	100·0
		102·4
		102·4
,,	23	101·0
		102·0
		102·4
,,	24	99·0
		101·0
		103·4
,,	25	99·4
		102·0
		105·0
,,	26	102·2
		104·3
		104·4
,,	27	102·2
		104·2
		102·2
,,	28	101·4
		104·2
		105·2
,,	29	99·3
		100·0
		99·1
		died

No. 193.

November	18	100·3
		101·2
,,	19	101·0
		100·3
		101·2
,,	20	100·2
		101·0
		102·1
,,	21	101·0
		102·0
		101·1
,,	22	101·1
		100·3
		101·0
,,	23	100·1
		100·0
		101·4
,,	24	99·4
		101·0
		101·1
,,	25	100·2
		101·1
		102·1
,,	26	100·1
		101·0
		101·3
,,	27	103·3
		105·0
		104·1
,,	28	103·0
		105·0
		105·2
,,	29	103·0
		104·4
		106·0
,,	30	103·0
		102·2
		106·0
December	1	105·0
		105·4
		105·0
,,	2	104·1
		103·4
		104·0
		died

APPENDIX 10.—STATEMENT OF VACCINES, ETC., ISSUED.

(i) STATEMENT OF VACCINE LYMPH SUPPLIED BY THE BACTERIOLOGICAL INSTITUTE, GRAHAM'S TOWN, DURING THE YEAR ENDING 31ST DECEMBER, 1897

(A) *To Governments outside the Colony.*

Basutoland	1,350
Orange Free State	350
South African Republic	1,450

(B) *To Resident Magistrates and District Surgeons in the Colony.*

	Tubes.
Aberdeen	45
Albany	19
Alexandria	100
Alice	6
Aliwal North	150
Barkly East	425
Barkly West	500
Bedford	10
Bredasdorp	37
Britstown	200
Burghersdorp	20
Butterworth	450
Cala	10
Caledon	50
Calitzdorp	62
Calvinia	48
Cape	1,865
Carnarvon	36
Cathcart	200
Clanwilliam	150
Colesberg	350
Cradock	36
Douglas	200
Durbanville	20
East London	24
Elliott	100
Elliotdale	210
Engcobo	110
Fort Beaufort	100
Frazerburg	154
George	850
Glen Grey	1,000
Graaff-Reinet	50
Griquatown	200
Herschel	110
Hopetown	80
Hopefield	25
Humansdorp	170
Jamestown	37
Jansenville	255
Kenhardt	140
Kentani	272
Kimberley	1,400
King William's Town	180
Knysna	68
Kokstad	1,300
Kuruman	500
Ladismith	32

	Tubes.		Tubes.
Lady Frere	800	Riversdale	12
Libode	1,650	Robertson	90
Lusikisiki	2,500	Rondebosch	50
Mafeking	50	Somerset West	100
Malmesbury	50	Springbokfontein	80
Middelburg	50	Sterkstroom	26
Montagu	78	Simon's Town	144
Mossel Bay	40	Steynsburg	60
Mount Ayliff	300	Stockenstroom	6
Mount Fletcher	9	Steytlerville	120
Mqanduli	124	Stutterheim	100
Namaqualand	50	Sutherland	36
Nqamakwe	1,375	Swellendam	25
Ngqelini	200	Tabankulu	500
Oudtshoorn	60	Tarka	25
Paarl	75	Tsolo	500
Pearston	48	Tsomo	300
Peddie	880	Tulbagh	12
Philipstown	110	Uitenhage	900
Piquetberg	228	Umtata	150
Port Alfred	20	Umzimkulu	2,120
Petrusville	25	Upington	200
Port Elizabeth	130	Van Rhynsdorp	24
Port Nolloth	100	Victoria West	50
Port St. John	400	Vryburg	300
Prieska	53	Willowmore	20
Prince Albert	140	Wodehouse	60
Queenstown	175	Woodstock	56
Qumbu	1,000	Wynberg	85

(C) *To Private Practitioners and Persons outside the Colony.*

	Tubes.		Tubes.
Orange Free State	3	Rhodesia	500
Natal	125	South African Republic	8,704

(D) *To Private Practitioners and Persons in the Colony.*

	Tubes.		Tubes.
Barkly East	10	Kimberley	148
Bredasdorp	25	King William's Town	15
Cape	1,056	Ladismith	15
De Aar	40	Mossel Bay	12
East London	6	Namaqualand	3
Graaff-Reinet	3	Port Elizabeth	200
Graham's Town	16	Queen's Town	2
Hanover	25	Riebeek West	12

SUMMARY.

	Tubes.	Needles.	£	s.	d.
(*a*) To Governments outside the Colony	3,150 at 1/6	—	236	5	0
(*b*) To Colonial Government ..	29,227 at 1/6	58 at 3d.	2,192	15	0*
(*c*) To Private Persons outside the Colony	9,332 at 1/6	—	699	18	0
(*d*) To Private Persons in the Colony	1,588 at 1/6	—	119	2	0
	43,297	58	3,248	0	0

* By special agreement a rebate is allowed on all Vaccine Lymph issued to the Department of the Honourable the Colonial Secretary.

(ii) Statement of Anthrax Vaccine issued from the Bacteriological Institute, Graham's Town. during the Year ended 31st December, 1897,

		Tubes.	Syringes			Tubes.	Syringes
1	Bloemfontein ..	5	0	10	King Wm's. Tn.	80	2
2	Bredasdorp ..	16	1	11	Langberg ..	60	2
3	Barkly West ..	4	0	12	Port Elizabeth ..	2	0
4	Douglas ..	368	17	13	Rhodesia ..	5	1
5	Ficksburg, O.F.S.	5	2	14	Taungs ..	23	0
6	Geluk, Vryburg	50	0	15	Umtata ..	60	1
7	Hay	370	28	16	Vryburg ..	11	0
8	Kimberley ..	344	3				
9	Kuruman ..	41	1			1,444	58

TOTAL:

					£	s.	d.
1,444 Tubes at 2/6..					£180	10	0
Syringes 2 at 14/-..	£1	8	0				
,, 1 at 15/-..	0	15	0				
,, 6 at 11/-..	3	6	0				
,, 49 at 7/6..	18	7	6		23	16	6
					£204	6	6

(iii) Statement of Mallein issued from the Bacteriological Institute, Graham's Town, during the Year ended 31st December, 19·7

1	Cape Town ..	5 bottles	2	Kimberley ..	1 bottle

TOTAL

6 Bottles at 2/6 each .. £0 15 0

(iv.).—Statement of Sponziekte Vaccine supplied by the Bacteriological Institute, Graham's Town, during the Year ended 31st December, 1897.

		Tubes.	Syringes			Tubes.	Syringes.
1	Albany	55	1	5	Middelburg ..	4	1
2	Alice	50	1	6	Pretoria ..	10	1
3	Heilbron, O.F.S.	19	1	7	Uitenhage ..	20	0
4	Ladybrand, O.F.S.	20	0				

Total :

	£	s.	d.
178 Tubes at 1s. per tube ..	£8	18	0
5 Syringes at 14s. each ..	3	10	0
	£12	8	0

(v.).—Statement of Diphtheria Anti-toxin and Syringes supplied from the Bacteriological Institute, Graham's Town, during the Year ended 31st December, 1897.

		Bottles.	Syringes.			Bottles.	Syringes.
1	Britstown ..	1	0	5	Robben Island	0	18
2	Johannesburg ..	6	0	6	Smithfield, O.F.S.	2	2
3	Kimberley ..	12	1	7	Somerset East	6	2
4	Nylstroom, O.F.S.	0	2				

Total :

	£	s.	d.
21 Bottles at 2s. 6d. each ..	£2	12	6
6 ,, 1s. 6d. ,, ..	0	9	0
25 Syringes at 14s. ,, ..	17	10	0
	£20	11	6

(vi.).—Statement of Lung-sick Virus and Inoculating Needles supplied by the Bacteriological Institute, Graham's Town, during the Year ended the 31st December, 1897.

		Tubes.	Needles.			Tubes.	Needles.
1	Alice	5	1	5	Mafeking ..	10	0
2	Barkly East ..	0	2	6	Nqamakwe ..	0	1
3	Graham's Town ..	0	2	7	Peddie.. ..	16	1
4	Lobatsie, British Prot.	6	0	8	Steynsburg ..	27	1
				9	Stutterheim ..	6	0

Total :

	£	s.	d.
70 Tubes at 1s. 3d. each ..	£4	7	6
8 Needles at 4s. 6d. each ..	1	16	0
	£6	3	6

(vii.).—STATEMENT OF LOCUST FUNGUS SUPPLIED FROM THE BACTERIOLOGICAL INSTITUTE, GRAHAM'S TOWN, DURING THE YEAR ENDED 31ST DECEMBE 1897.

		Tubes			Tubes.
1	Alexandria	6	7	India	10
2	Argentine Republic	162	8	Kokstad	3
3	Coega	2	9	Ngqeleni	100
4	East London ..	72	10	Rhodesia	2,195
5	Graham's Town ..	2	11	Uitenhage ..	2
6	Idutywa	5			

Total .. .. 2,559 tubes.

Money received £40 12 4

(viii.).—STATEMENT OF BACTERIOLOGICAL EXAMINATIONS MADE DURING THE YEAR ENDED 31ST DECEMBER, 1897.

For Private Persons outside the Institute.

(a) DIPHTHERITIC.

No.	Date.	Locality from which case forwarded.
1	March 27	Rondebosch
2	June 29	Kimberley
3	August 25	King William's Town
4	Sept. 15	Queen's Town
5	Oct. 25	Mowbray

(b) SPUTUM.

No.	Date.		Locality from which specimen forwarded
1	January	5	Port Elizabeth
2		11	,,
3	February	10	Cape Town
4		13	Graham's Town
5	March	2	James Town
6		2	Johannesburg
7		6	Graham's Town
8		9	,,
9		13	Ceres
10	April	2	Aliwal North
11		2	,,
12		28	,,
13	May	4	Graham's Town
14		26	Rondebosch
15	June	3	Graaff-Reinet
16		8	Middelburg
17	July	21	,,
18	August	25	Fort Beaufort
19		26	Knysna
20	September	29	Graham's Town
21	October	25	Hanover
22		28	Graham's Town
23	November	4	,,
24		4	Fort Beaufort
25	December	11	Mowbray
26		13	Graham's Town
27		16	Queen's Town
28		28	Aliwal North.

(*c*) URINE.

No.	Date.		Locality from which specimen forwarded.
1-6	February	10 (6)	Johannesburg
7	March	2	Middelburg
8		12	Graham's Town
9		20	,, ,,
10	April	12	,, ,,
11	June	15	Middelburg
12	August	11	Pietermaritzburg
13	November	5	Uitenhage
14	December	23	,,

(*d*) TUMOURS.

No.	Date.		Locality from which specimen forwarded.
1	March	16	Murraysburg
2		25	Graham's Town
3		29	,, ,,
4	April	12	,, ,,
5	May	9	,, ,,
6	June	30	Mowbray
7	July	21	Gordonia
8	August	24	Claremont
9	September	29	Cape Town

(*e*) WATER.

No.	Date.		Locality from which specimen forwarded.
1-3	November	5	Queen's Town
4-6	December	3	,, ,,

(*f*) FŒCES.

No.	Date.		Locality from which specimen forwarded.
1	March	2	Somerset East
2		13	,, ,,
3	April	12	Graham's Town
4		12	,, ,,
5	October	19	Queen's Town

(*g*) BLOOD.

No.	Date.		Locality from which specimen forwarded.
1	March	22	Graham's Town
2	April	8	,, ,,

(*h*) INTERNAL ORGANS.

No.	Date.		Locality from which specimen forwarded.
1	December	23	Port Elizabeth

SUMMARY.

Nature of Examination.	Number.	£	s.	d.
Diphtheria	5	1	7	6
Sputum	28	7	0	0
Urine	14	3	10	0
Tumours	9	4	10	0
Water	6	3	3	0
Fœces	5	2	10	0
Blood	2	0	10	0
	1	3	3	0
Total	70	£25	13	6

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CAPE OF GOOD HOPE.

COLONIAL SECRETARY'S MINISTERIAL
DIVISION.

REPORT

OF THE

DIRECTOR

OF THE

COLONIAL BACTERIOLOGICAL INSTITUTE.

For the Year 1897.

Presented to both Houses of Parliament by command of His Excellency the Governor

1898.

CAPE TOWN:
W. A. RICHARDS & SONS, GOVERNMENT PRINTERS
1898.
A207 [illegible]

[G. 24—'98.]